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TRAUMATIC ARTERIAL VASOSPASM

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BOSTON

THE opportunity of treating a large number of war wounds of the extremities has demonstrated that vasospasm, of varying intensity and obstinacy, often complicates these injuries. This paper is not primarily concerned with cases of subtotal ischemia in which the immediate viability of the part is threatened but with the less dramatic and much larger group in which the persistence of a relatively low-grade arterial spasm is likely to cause nutritional changes adversely affecting the functional result.

Everyone is alarmed when a wound involves or suggests injury to a major artery; modern surgeons are equally apprehensive about the cold, pulseless limb produced by a widespread and intense arterial spasm, of which there have been many reports in recent years. Too little concern, however, is generally shown about the more moderate picture of arterial spasm, which holds little or no threat to the viability of the limb but a serious one to the ultimate function. In my opinion some of these unsatisfactory functional results are not entirely unavoidable. They have been explained by diagnoses of traumatic arthritis, Sudek's atrophy, neurotrophic edema and other titles to fit the prominence of certain signs found in the so-called "causalgic states," and thus the surgeon has considered himself absolved of responsibility for the functional result. An impaired arterial blood supply due to a subacute arterial spasm is regarded as the fundamental cause of much disability not inherent in the original injury. Recognition of the ischemia and rational specific therapy are essential to proper treatment.

INCIDENCE AND PREDISPOSING FACTORS

Although actual percentage figures of the occurrence of vasospasm in war injuries are not available, the clinical experience of one particularly interested in this phenomenon is not without value. In a hospital averaging 1500 war casualties, of which about 85 per cent were surgical, and excluding cases of trench foot (all of which presented some degree of vasospasm at one stage of the disease), an average of 12 to 15 cases were constantly being watched and treated for vasospasm that was considered sufficiently pronounced to endanger the functional result.

From this experience, can any conclusion be reached regarding what sort of injury is likely to be complicated by arterial spasm? Are the increased wartime incidence and interest in this and allied disorders due to anything peculiar to battle casualties, and what application is there to civil practice? These questions are considered under the following headings: the extent and location of the initial injury; the tissues involved; individual susceptibility; and blast effect.

Extent and Location of Original Injury

Contrary to what might be expected, the incidence of arterial spasm does not parallel the severity of the original wound. Patients with extensive wounds involving shattering compound fractures, with much soft-tissue destruction of the upper portion of an extremity, may have quite adequate circulation in the distal portion of the same limb, whereas minor injuries to toes or fingers may set up a vasospasm so severe as to threaten function. Wounds were often seen with such extensive loss of substance as to make one wonder how sufficient circulation could get through to the distal portion, and yet the circulation there was found to be adequate. In 1 case, in spite of extensive loss of substance in a wound of the elbow, no clinical evidence of vasospasm appeared, and the circulation to the distal portion of the extremity remained adequate (Fig. 1).

In another case a shell fragment tore through the upper extremity, taking with it the greater portion of the humerus (Fig. 2). In spite of the soft-tissue destruction obviously involved, the circulation to the forearm and hand remained adequate. It should be noted that in these extensive injuries the wound is wide open — thoroughly decompressed. This factor is discussed below.

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Conversely, some of the most stubborn cases of vasospasm complicated mild injuries. A patient whose only injury was a bullet wound through the proximal phalanx of the left little finger that healed promptly, but with some deformity and a tender scar, developed such persistent vasospasm that dorsal ganglionectomy was required after all other

FIGURE 1. *Photograph of a Shell-Fragment Wound of the Right Arm.*

Despite extensive soft-tissue injury, circulation remained adequate.

efforts to restore circulation had failed. After this operation good circulation was restored, and the patient regained full function of the hand and fingers. This case is reported in detail below. Another patient was injured by a small shell fragment, which passed between the first and second toes of the left foot, causing mild wounds to the adjacent sides of the two digits. A week later the wounds were healed, but the foot was cold and cyanotic. So obstinate was the vasospasm that after all measures for vasodilatation, including repeated sympathetic blocks, had given only temporary relief, lumbar ganglionectomy was done two and a half months after injury, and the arterial supply subsequently remained adequate.

My experience is that the incidence of vasospasm does not rise with the severity of the wound but is found oftener in the smaller, quickly closed wounds than in the larger, wide-open ones. This suggests that tension is a factor and may explain the fact that a certain measure of success attended the decompression of early Volkmann's contracture by multiple, long incisions in the period when that complication was attributed to venous congestion. Although I am convinced that the proximate mechanism of Volkmann's contracture is an intense arterial spasm,[1] increased tissue tension may be a factor in the spasm.

Tissues Involved

Most people associate arterial spasm with injury to arteries or nerves. It is known that injured arteries have a natural tendency to contract to prevent excessive blood loss and that vasospasm is produced by the sympathetic system. It is logical, therefore, to look for direct trauma to large arteries or nerves in attempting to analyze the cause of vasospasm. Many reported cases with direct trauma, particularly to large arteries, have been complicated by extensive spasm. This is not, however, an essential part of the mechanism, and in the cases presented below, there was often no evidence of direct injury to main arteries or nerves. Illustrative of this are the 2 cases in which the injuries were confined to digits, with no possibility of direct trauma to important arteries or nerves but which presented stubborn vasospasm. In other cases, although the site of the wounds did

FIGURE 2. *Roentgenogram of a Shell-Fragment Wound of the Shoulder and Arm.*

Despite extensive loss of tissue, including the greater part of the humerus, circulation to the forearm remained adequate.

not offer such absolute preclusion of artery or nerve injury, evidence in favor of such injury was not usually found. Wounds from flying shell fragments followed no pattern and were seen in all locations, courses and degrees of severity — the impression that such factors influenced the incidence of vasospasm was not gained.

Individual Susceptibility

In an analysis of this sort, a predisposition of the patient must be considered at least briefly. There is

something to be said for such a possibility. Some people, even in the best of health, often suffer from cold extremities due to vasomotor lability. If to this is added the rigors of battlefront conditions before injury — physical and nervous exhaustion, deficiencies of diet and body hygiene and all grades of exposure to the elements — some element of predisposition cannot be denied.

Blast Effect

So far, rather negative conclusions have been reached, but one factor that may for the moment be called the "blast effect" is, in my opinion, positive. An appreciable number of self-inflicted wounds were treated. In these cases the first and second toes of the left foot were most frequently injured. Occasionally, a more determined or a less careful patient had blasted through the metatarsal region. Since these patients were usually in no particular hurry to be rehabilitated, their slow progress was at first attributed to lack of co-operation in active exercises. It was soon realized that in this type of wound the incidence of vasospasm was high — in fact, to some degree at least, almost universal.

In such close-range wounds the blast effect or concussion was obviously severe, with diffuse intracellular damage. Vasospasm has been noted in the so-called "crush syndrome" (Bywaters[2]). In fact, in a case in which the leg had been crushed in an air raid, vasospasm of the opposite, uninjured leg resulted in the loss of two toes.[3] This mechanism may be operative to some extent in so-called "wringer injury."

On the basis of the high incidence of vasospasm in cases in which the blast factor is heavy, it can perhaps be understood why war injuries, which are largely caused by ragged, irregular shell fragments propelled by high-explosive forces, are more frequently complicated by vasospasm than civil accidents. In 1935 Montgomery and Ireland[4] published a review of the literature in which they reported 44 cases of "traumatic segmentary arterial spasm" resulting from all kinds of trauma, of which 26 were the result of gunshot or high-explosive injuries. The comparatively smaller penetrating or perforating wounds probably cause more blast effect, at least on the remaining tissue, than the larger evulsing wounds, which are also decompressing.

In my experience the incidence of vasospasm is not dependent on the extent or location of the injury, or particularly on the structures involved; it is higher in the less open wounds, some predisposition may be operative, and diffuse tissue damage due to the concussive effect of explosive missiles is a factor.

Clinical Picture

In traumatized extremities vasospasm has been found in all degrees of intensity and distribution. This paper is concerned particularly with the less severe grades that might escape the unobservant. An example of arterial spasm in its simplest form occurred in a soldier who had suffered a rather minor soft-tissue wound over the proximal portion of the dorsum of the right foot that had nearly healed. Inspection of the foot revealed a striking and most unusual color change. The medial half of the dorsum of the foot, including the first and second toes, was blanched to a dead white, in contrast with the normal pink of the lateral half and the fourth and fifth toes — the third toe showed partial

FIGURE 3. *Photograph Illustrating Vasospastic Ischemia Secondary to a Penetrating Wound of the Calf.*
The three white areas are finger pressure spots. Note the irregular blanching, particularly in the distal portion of the foot.

changes. On palpation the blanched area was cold, and the pink area was warm; tactile sensation was less acute in the affected portion. The extremity constituted almost an arteriogram of occlusion of the dorsalis pedis artery — substantiated by an absent pulse in that vessel. The ward officer and the patient had noticed the condition for at least two or three days but had not been aware of any difficulty before that time. Treatment of any sort was postponed so that pictures could be made to record such a precisely demarcated example of arterial block, but they were never made. The next morning the foot was quite normal, and no recurrence took place — it was not possible to stimulate one even for the record. There was no intrinsic damage to the dorsalis pedis artery, which had been caught in a segmentary reflex spasm stimulated by some impulse from the nearby injury. Although most complete and sharply defined while in effect, it was

transient, and its spontaneous disappearance was equally complete. This case is not regarded as involving any threat to the ultimate function of this particular foot but rather as a classic example of arterial spasm in its simplest form. It is the element of which the more extensive and more obstinate syndromes are composed. The prominent features of such a picture are of interest.

Color change is the outstanding sign. It varies under different conditions but always differs from that in the opposite member. At room temperature and at rest the color is a combination of pallor and cyanosis (Fig. 3). The pallor is usually spotty, whereas the cyanosis is diffuse, increasing distally in depth. As compared to the opposite side the lack of red tones is distinctive. With dependency or exposure to cold, particularly on wet days out of doors, the cyanosis increases, and with elevation it fades and pallor increases. The extremity actually looks cold.

The surface temperature is obviously decreased, being lowest distally. Sometimes the patient volunteers this as a subjective complaint. The lower surface temperature is easily noticed by a comparison of palpation with that on the other side or the proximal part of the same extremity. All grades from a barely perceptible difference to a definite chill have been observed. Almost a part of the cool sensation is that of dampness — a combination sufficiently unpleasant to arrest attention and interest.

Swelling is generally seen in all injuries, particularly on use and dependency after plaster immobilization. Cases presenting ischemia are no exception. Anoxemia increases permeability of the capillary walls and increases interstitial fluid. The amount of fluid thus caused, however, is not large, and unless other factors contribute, edema is moderate.

An interesting group of symptoms is that based on the common denominator of local discomfort. Unlike the complaints in the true causalgias, severe pain is rare, although moderate degrees of pain may occur. The patient often complains of numbness, coldness, tingling or other such descriptive paresthesias but is reasonably comfortable so long as the extremity is supported and at rest. There is local tenderness, out of proportion, it may seem, to the force of palpation — indeed, even the threat of palpation is poorly received. The patient's guard is always up — a picket line that ward attendants and fellow patients soon learn not to cross. The extremity is held immobile, with the joints flexed. There is a disinclination to exercise and a disapproval of any manipulation. This symptom complex in itself retards rehabilitation and may make one suspect the patient of poor co-operation.

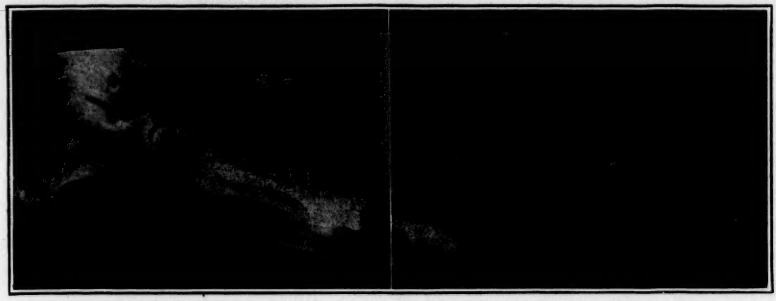

FIGURE 4. *Roentgenograms of Both Feet of a Patient with a Compound Fracture of Both Bones of the Right Lower Leg.*

This patient had signs of vasospastic ischemia when seen five months after injury. Note the osteoporosis in the bones of the right foot (right).

Finally, the most specific, reliable and convincing sign of arterial spasm is the finding of diminished or absent peripheral pulses, complemented by adequate pulses on the alternate side and variably responsive to measures of vasodilatation. The parallel between the observable evidence of ischemia and the lowered pulse volume is reasonably consistent.

VASOSPASM AS A SPECIFIC ENTITY

Of course, vasospasm is not accountable for all circulatory deficiencies following injury. Swelling and cyanosis are usual after fractures, particularly during the initial mobilization period. Impaired venous return and the softening of disuse and lowered local metabolism must be overcome in all injuries of any moment. Beyond this, however, the careful observer will find that in some cases a definite deficiency exists on the arterial side; the swelling is less prominent, the cyanosis contains more pallor and less redness, the surface temperature is lower,

there is more discomfort, and the distal pulse is diminished or absent. Although arterial spasm probably occurs oftener in war wounds it seems to complicate certain civil injuries more frequently than may have been suspected. All grades of severity and of extension are possible, and the development can be insidious. Arteriospasm has probably been overlooked at times, or its results attributed to other causes.

Blackwood[5] presents the following description:

> The arteries are the supply lines. An ischemic limb resembles the inhabitants of a beleagered town. With supplies diminished or cut off they can keep up normal appearances for a time, but soon they begin to starve. They feel cold, their faces are pale or blue, they become less active and as conditions grow worse they become apathetic, or complain loudly to those in authority. In the human limb there is absence of arterial pulsations, lowering of surface temperature, pallor or cyanosis, anaesthesias and pain.

As stated above, this report is not primarily concerned with the limbs whose lines have been completely cut off but rather with those that are on short rations. Malnutrition, which is the basic difficulty, results in degenerative changes of the musculoskeletal system primarily. Muscle atrophy, osteoporosis, limited and painful motion, flexion deformities and joint stiffness are the price of a prolonged arterial inadequacy (Fig. 4 and 5). A rich blood supply is of primary importance for repair of injury and for maintaining good tone of the extremity pending return of function. Arterial spasm complicating injury throttles a blood supply that needs to be at its best.

Therapy

So far as possible, cases presenting vasospastic ischemia were grouped together. It was believed that both the patients and the ward personnel could more easily be indoctrinated in the general program and the results better evaluated. Basically, blood loss and dehydration were corrected, and adequate diet, added vitamins and sufficient rest assured. Some patients received 4 gm. of aspirin a day and 30 cc. of whisky three times a day, but no improvement was noted from this medication. Buerger's exercises were used routinely, without relief of the arterial spasm. The extremities were kept warm but not overheated (these patients were not allowed beds near the stoves), and exposure to cold and inclement weather was avoided. Heaters to other parts of the body were used, and warm (not hot) soaks were given to the affected parts several times a day.

Paravertebral novocain blocks, which were employed as soon as the condition was observed, al-

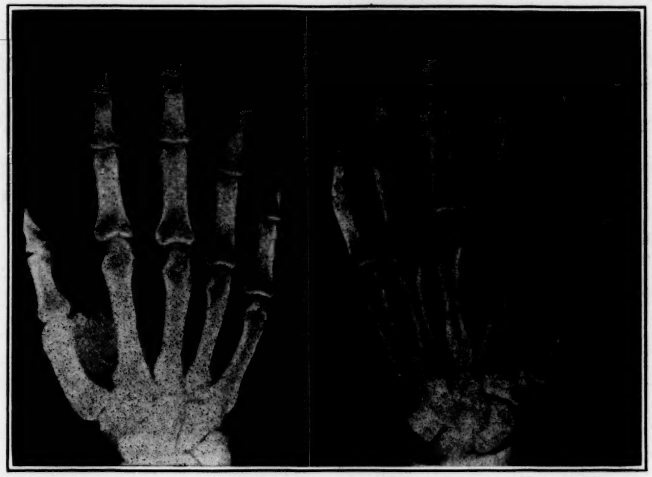

Figure 5. *Roentgenograms of Both Hands Taken Three Months after the Patient Sustained a Shell-Fragment Wound of the Upper Right Arm, with No Fracture or Nerve Injury.*

The hand was still cold and blue. Note the decalcification, the haziness of joints, the atrophy of the soft parts and the flexion of fingers of the right hand (right) as compared with those of the normal hand (left).

most universally caused improvement for at least a short time — even if only for a couple of hours. More frequently, such improvement lasted for two or three days. Blocks were repeated several times if necessary and so long as improvement increased

FIGURE 6. *Photograph of Both Hands of a Patient Presenting Vasospastic Ischemia (Case 1).*

Note the moderate swelling and cyanosis of the right hand, which was also cool.

with successive blocks. If the interval of improvement decreased progressively, ganglionectomy was considered. This operation was done in 4 cases in which vasospasm persisted beyond all the treatment

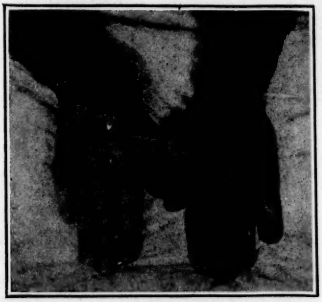

FIGURE 7. *Photograph of the Same Hands after Right Upper Dorsal Ganglionectomy.*

The cyanosis and swelling have disappeared. After initial marked vasodilatation, the hand took on a normal color.

mentioned above, and in each case permanent relief of symptoms resulted.

CASE REPORTS

The following cases of persistent low-grade vasospasm illustrate the difficulties sometimes encountered.

CASE 1. J. C., a private first class, while in close fighting in France on June 14, 1944, sustained a bullet wound of the right fifth finger and was struck on the forehead by the stock of an enemy rifle. He was not unconscious. On the same day he was admitted to a field hospital, where the finger wound was débrided. He was evacuated by air to England, arriving at a general hospital on June 17. At that time a lacerated wound in the center of the forehead was found; x-ray examination of the skull was negative. A perforating rifle wound, whose entrance was on the dorsum of the proximal phalanx of the right fifth finger and whose exit was on the palmar aspect of the same phalanx, was also observed, with a compound, comminuted fracture of the proximal phalanx.

The wound was dressed with vaseline-soaked gauze, and a splint was applied. By July 11 the wounds had healed, and the fracture had united solidly; the finger was stiff, tender

FIGURE 8. *Photograph of Both Feet of a Patient with Shell-Fragment Wounds of the First and Second Toes of the Right Foot, Complicated by Obstinate Vasospastic Ischemia (Case 2).*

Note the moderate swelling, as well as the diffuse cyanosis contrasting with the finger-point blanching.

and painful, however. The patient was sent to the physiotherapy section, where he received daily whirlpool-bath treatments and exercises for 2 weeks. On August 4 he was transferred to the rehabilitation group, where for 1 month he took part in the program but complained of pain and tenderness in the finger and that the whole hand became blue and cold, particularly when he was outdoors. Because of these complaints he was given further physiotherapy, with no improvement, and was returned to the hospital on September 4. Examination disclosed general pallor of the hand, with cyanosis (Fig. 6). The radial pulse was palpable, but the ulnar pulse was not. A stellate ganglion block with novocain was done on September 6. This resulted in a warm, pink hand with palpable pulses that lasted for 3 days. Owing to a novocain reaction the block was not repeated, and on September 11 the finger scar was revised and a spicule of bone was removed in the hope of relieving pain. At the same time the ulnar artery was stripped just above the wrist. Good pulsations, surface color and temperature followed this

procedure, but after 1 week the signs and symptoms of local ischemia returned. By that time even the uninjured fingers had limited motion and the functional result was poor.

On September 28 an upper dorsal ganglionectomy was done through a posterior incision, the second and third thoracic segments being removed. This resulted in a warm, pink hand with good pulses (Fig. 7), and no further evidence of ischemia. Function of the hand was completely restored except for permanent stiffness of the proximal joint of the little finger.

CASE 2. W. S., a 31-year-old private, was wounded in action in France on July 7, 1944, by fragments from an 88-mm. shell. He sustained wounds of the right upper arm, both buttocks and the first two toes of the right foot. On the same day the wounds were débrided, and a metallic foreign body was removed from the second toe under Pentothal anesthesia at an evacuation hospital.

On July 13 the patient arrived at a general hospital in England, where examination revealed a moderate penetrating wound on the posterior aspect of the right upper arm, moderate lacerating wounds of both buttocks, moderate penetrating wounds on the medial aspect of the right great toe and the lateral aspect of the right second toe, compound, comminuted fractures of the middle and distal phalanges of the right first toe and middle phalanx of the second toe, and a metallic foreign body in the middle phalanx of the right second toe.

The wounds healed promptly without surgical closure. The patient was urged to get about but complained that pain in the great toe prevented his becoming ambulatory. On August 15 the ward surgeon insisted that he attempt to walk. When he did so the foot became swollen, cyanotic and cold. He complained of pain throughout the whole foot, particularly over the scar on the great toe. The dorsalis pedis pulse was palpable, but weaker than that of the opposite foot. A course of physiotherapy was ordered, with no improvement in 2 weeks. On September 1 a plastic repair was done on the scar of the first toe, and the foreign body was removed from the second toe. The scar subsequently gave no further pain, but the foot remained cold and cyanotic and he complained of pain through the foot (Fig. 8).

On September 13 a lumbar ganglion block was done, with good immediate results. The color and surface temperature improved, and there was subjective relief that lasted for 4 days. On September 20 a second lumbar block afforded similar relief but for only for 1 day.

On September 28 a lumbar ganglionectomy was done, with sustained relief. Thereafter, the foot remained warm, pink and painless. There was some swelling when the patient became ambulatory, but this subsided with use and the patient was walking well and without complaint when he was discharged from the hospital on November 2 (Fig. 9).

These cases, which are extreme rather than representative, are reported to illustrate the full range of therapy, of which lesser measures usually suffice. In most cases careful attention to supportive routine, in addition to several sympathetic blocks, restored adequate circulation. Often, a single novocain block brought about improvement that could be maintained by careful attention to the supportive measures mentioned above. The amount of treatment indicated depends entirely on the stubbornness of the vasospasm, which can be measured only by its response to therapy.

CIRCULATION

In a discussion of the treatment of vasospasm some consideration should be given to the objections to sympathetic paralysis recently presented by Cohen[6, 7] and supported by the experiments of Friedländer et al.[8] Cohen points out that the circulation of the deep structures, particularly the muscle bellies, is independently regulated as compared with that of the surface structures, because their needs for increased blood supply do not coincide. He concludes that an extremity with a blanched, cool surface represents a protective mechanism, the available blood being shunted to the deep structures, which are more vulnerable to its loss. He believes that sympathetic paralysis, under these conditions, diverts blood to the surface

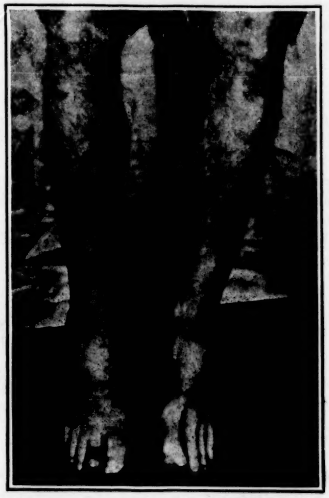

FIGURE 9. *Photograph of the Same Feet after Right Lumbar Ganglionectomy.*
Note the improvement in color and the absence of swelling.

at the expense of the deep circulation and thus prevents the anticipated result.

These arguments, besides being of considerable interest, are of more than academic importance to anyone responsible for the care of this complication. Obviously, the treatment will be almost exactly reversed depending on which view is favored. These reports, which appeared while many of these patients were under treatment, afforded no comfort. In spite of a revaluation of results with these arguments in mind it could be concluded only that extremities presenting varying degrees and extent of vasospasm were clinically improved by sympathetic paralysis. Not only were surface changes noted but also pulses improved, discomfort decreased and activity increased. The treatment was therefore continued, pending further study. The relation of the deep and surface circulations will stand further investigation, but a reversal of the treatment of vasospasm does not seem to be indicated.

Clinically, the indication for sympathetic paralysis is the presence of vasospasm — in fact, sympathetic

block is a therapeutic test for the presence of vasospasm as distinguished from mechanical occlusion. If, in an ischemic extremity, one could be certain that the impairment was organic and fixed with no spasm of the collateral vessels, there would be no point in attempting to alter the distribution of this fixed supply. Clinical improvement following sympathetic block is believed to indicate the presence of vasospasm, and there is increasing evidence that it complicates peripheral injury and disease more frequently than has been appreciated.

Summary

Vasospasm of varying severity and obstinacy may complicate peripheral injuries, particularly those in which there is an element of concussion involving increased tissue tension.

Neither extensive injury nor involvement of nerves or blood vessels is necessary to the development of vasospasm.

Subjectively, there is local discomfort and resistance to function. Objectively, there is decreased surface temperature, blotchy cyanosis and diminished pulses.

A persisting, low-grade ischemia causes trophic changes that materially impair the functional result.

Two cases of traumatic arterial vasospasm in which sympathetic block resulted in clinical improvement are presented.

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VAGINAL SMEARS AS AN AID IN THE DIAGNOSIS OF EARLY CARCINOMA OF THE CERVIX*

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Of the 17,000 annual deaths in the United States from uterine cancer the large majority are caused by carcinoma of the cervix. It has been stated that only 11 per cent of cervical cancers reach the surgeon in the early stage, and 60 per cent are completely inoperable when treatment is begun.[1] There is still an average delay of seven months from the onset of symptoms to the institution of treatment. Efforts to educate physicians and the public to the vital importance of immediate diagnosis and treatment at the onset of abnormal vaginal bleeding or discharge have been only partially successful. Moreover, even clinically early cases continue to show recurrence of the disease after operation or radiation.

It is possible that the entire concept of the life history of cervical cancer needs revision: that the so-called "early case" presenting a short history of bleeding or a minimal visible lesion is, in fact, already a moderately advanced or advanced cancer. It is possible that early cancer causes no symptoms and that diagnosis in the incipient stage would be followed in most cases by cure of the disease.

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This hypothesis is substantiated by two recent reports. In July, 1946, Pund and Auerbach[2] published a study of 1200 cases in which the cervix was removed by hysterectomy for conditions other than cancer. Preinvasive cancer was found to be present in 47 cases (3.9 per cent); in addition several cases of unsuspected invasive cancer were found. The presence of cancer was not recognized from the gross appearance of the cervix in a single case. *Only one out of 4 patients had had abnormal bleeding.* The average age of the 47 women showing preinvasive cancer was thirty-six and a half years, in contrast to a second group of 50 consecutive biopsies showing invasive cancer, in which the average age was forty-eight and a half years. TeLinde[3] found identical figures — a thirty-six-year average age for 11 cases of preinvasive as contrasted with an average age of forty-eight years for overt cervical cancer.

In September, 1946, Taylor and Guyer[4] reported a case in which a biopsy of the cervix, taken in 1938, had been misdiagnosed. One pathologist reported epithelioma, transitional type, but he was overruled by a second pathologist, who believed that the tissue was insufficient for diagnosis. No operation was done. The patient had been examined every six months for seven years without the detection of any abnormality of the cervix. In 1945 a proliferative nodular lesion was seen on the cervix, a biopsy from

which showed early squamous-cell carcinoma with invasion. Preparation of additional sections from the tissue obtained seven years previously showed not only that cancer had been present but that (as in TeLinde's cases) beginning invasion had occurred in certain areas. The patient had been harboring cancer of the cervix without symptoms and without clinical evidence of the disease for seven years, and the clinical diagnosis seven years later was still "early carcinoma." Taylor and Guyer refer to Stevenson's[5] cases, one developing "clinical cancer" eight and a half years and another showing microscopical invasion three years after previous biopsies had demonstrated noninvasive carcinoma of the cervix. Smith and Pemberton[6] found an interval of four to six years between the discovery of early lesions by biopsy and the development of clinical cancer.

Rubin[7] reports 2 cases of early squamous-cell carcinoma of the cervix. One patient was followed for seven years subsequent to a positive biopsy, with no symptom or sign referable to the malignant lesion. The other patient showed no clinical evidence of disease until five years after the original positive biopsy.

If there is a period of years before the appearance of symptoms during which cancer of the cervix is nevertheless present and capable of diagnosis by serial section of the excised cervix, there exists, if one could but make use of it, a critical period during which treatment should result in cure in all or in a high percentage of cases. Although such lesions are often detected by biopsy, a satisfactory biopsy specimen is difficult to obtain in early cases in which no suspicious area is visible or in which, as occurred twice in our series, the lesion is endocervical. The pathologist can report cancer only if an area containing cancer is provided him.

In 1941 Papanicolaou and Traut[8] described a new method for the diagnosis of uterine cancer. Technical details of this test are described in their publication, as well as in a previous paper from this laboratory.[9] In brief, a dry pipette with capillary opening and capped by a rubber suction bulb is inserted into the posterior vaginal fornix; the bulb is released, and the pipette withdrawn. Fluid so obtained is blown upon a glass slide, fixed while still wet in equal parts of ether and 95 per cent alcohol and later stained and examined for cancer cells. Criteria for the recognition of cancer cells are given in the two publications referred to above.

Papanicolaou,[10] in 1943, reported 7 cases of "intradermal" carcinoma of the cervix in which vaginal smears were positive for cancer. Ayre[11] described 19 cases in which the interesting diagnosis of "precancer" was made. Abnormal cells were found in the cervical secretions of these women. It is difficult to know whether these cases fall into the group discussed below. Hertig[12] reports 8 positive and 5 negative smears in 13 cases of cervical carcinoma in situ.

In 1945 Meigs et al.[13] reported 3 cases of cervical carcinoma in situ in which vaginal smears were positive. Meigs[14] reported from this laboratory 2 additional cases of preinvasive carcinoma of the cervix in which vaginal smears were positive. In the 12 cases of cervical carcinoma in situ reported below, vaginal smears were positive in 10.

In the total group of 17 cases, 14 patients gave a history of bleeding or discharge, and 3 were without symptoms — of these, 2 had positive vaginal smears (Table 1). Initial biopsies were positive in 4 cases, and in 2 the initial biopsy was reported "insufficient for diagnosis" — in 1 of these a second biopsy showed carcinoma in situ. The biopsy diagnosis was chronic cervicitis in 5 cases and leukoplakia in 1. No biopsy

TABLE 1. *Pertinent Data in Cases of Cervical Carcinoma.*

Case No.	Age	Symptoms	Vaginal Smear	Biopsy	Pathological Diagnosis
	yr.				
1*	46	Occasional bleeding for 3 mo.	Positive	—	Carcinoma in situ
2*	48	Vaginal spotting for 1 wk.	Positive	Insufficient for diagnosis	Carcinoma in situ
3*	49	Spotting for 2 mo.	Positive	? Carcinoma in situ	Carcinoma in situ
4†	35	Spotting for 6 wk.	Positive	Chronic endocervicitis	Carcinoma in situ
5†	45	None	Positive	Chronic endocervicitis	Carcinoma in situ
6	71	None	Positive	—	Carcinoma in situ
7	42	Spotting	Positive	Carcinoma in situ	—
8	43	Postcoital spotting	Positive	—	Carcinoma in situ
9	63	Bleeding for 2 mo.	Positive	1st — insufficient for diagnosis; 2nd — carcinoma in situ.	Carcinoma (? in situ)
10	57	Spotting for 8 mo.	1st — unsatisfactory; 2nd — positive.	—	Carcinoma in situ
11	38	Precancerous lesion suspected by physician	Negative	Chronic endocervicitis	Carcinoma in situ
12	39	Staining for 10 days	Positive	Carcinoma in situ	Carcinoma in situ (? early invasion)
13	38	Occasional spotting	Positive	No specimen obtainable	Carcinoma in situ
14	57	Increase in discharge for 3 months; no bleeding.	Positive	Carcinoma in situ	—
15	49	Bleeding for 1 day	Positive	Chronic endocervicitis	Carcinoma in situ
16	42	None	Positive	Leukoplakia	Carcinoma in situ
17	31	Bleeding for 4 mo.	Negative	Chronic endocervicitis	Carcinoma in situ

*Previously reported by Meigs et al.[13]
†Previously reported by Meigs.[14]

was performed in 4 cases, and in another no biopsy specimen could be obtained. Thus, in 8 of 12 cases, the initial biopsy failed to disclose the presence of carcinoma in situ, whereas of 17 cases examined by vaginal smear, this test was positive in 15.*

The following are brief abstracts of the last 12 cases.

CASE 6. H. P. (private case), a 71-year-old woman whose cervix had been repaired in 1920, had been given radium for flowing in 1923. There had been no hot flashes or other symptoms. Examination disclosed scarring of the cervix. Biopsy revealed no suspicious area. Vaginal smears on February 12 and June 4, 1945, were positive. A hysterectomy was performed on July 24 and at the midpoint in the cervical canal a polyp, 0.5 by 0.3 cm. in diameter, was found. The pathological diagnosis was epidermoid carcinoma in situ.

CASE 7. S. D. (M.G.H. 503466), a 42-year-old woman, complained of spotting preceding her periods. Examination disclosed an area of erosion about the os, with some bleeding at the lower margin. No suspicious area was seen. A vaginal smear was positive. The pathological diagnosis on a biopsy specimen was epidermoid carcinoma in situ.

CASE 8. R. B. (M.G.H. 547504), a 43-year-old woman, had had a supracervical hysterectomy for menorrhagia of 2 years' duration 5½ months previously. She complained of severe hot flashes. Vaginal spotting followed coitus 1½ months after operation. A vaginal smear was positive. No biopsy was performed. The pathological diagnosis was epidermoid cervical carcinoma in situ.

CASE 9. S. S. (M.G.H. 502116), a 63-year-old woman, had reached the menopause 15 years before admission. She had had no hot flashes. Bleeding for 1 day had occurred in June, 1945, and for 3 days in July. At biopsy on August 31 the tissue was insufficient for diagnosis; in another one, taken on September 7, the pathological diagnosis was epidermoid carcinoma of the cervix in situ. A vaginal smear on September 9 was positive. The final pathological diagnosis was epidermoid cervical carcinoma (? in situ).

CASE 10. A. H. (M.G.H. 519079), a 57-year-old woman who had reached the menopause 4 years previously, complained of occasional vaginal spotting of 8 months' duration until a curettage in another hospital 1 month before admission. A vaginal smear in January, 1946, was unsatisfactory; a smear on February 5 was positive. The cervix appeared normal. No biopsy was performed. The final pathological diagnosis was epidermoid cervical carcinoma in situ.

CASE 11. A. R. (M.G.H. 457627), a 38-year-old woman, had been informed of a precancerous lesion of the cervix by an Army medical officer on June 20, 1944. For 10 years she had noticed bleeding and pain after intercourse. A vaginal smear on August 5 was negative. A biopsy on August 7 revealed chronic endocervicitis. The pathological diagnosis on the excised uterus was epidermoid cervical carcinoma in situ.

CASE 12. M. D. (private case†), a 39-year-old woman, complained of staining for 10 days. Biopsies on September 28 and October 6, 1944, revealed carcinoma in situ. A vaginal smear on October 24 was positive. The pathological diagnosis was squamous-cell cervical carcinoma in situ (? early invasion).

CASE 13. K. W. (private case), a 38-year-old woman, had had regular periods but intermenstrual bleeding for 3 months. The cervix showed a small area of leukoplakia. No tissue was obtainable for biopsy. A vaginal smear on November 12, 1946, was positive. The pathological diagnosis was epidermoid cervical carcinoma in situ.

*A slide in another case was sent to this laboratory from the Free Hospital for Women. The smear was thought to be negative, although a pathological diagnosis of cervical carcinoma in situ had been made.

†Reported through the courtesy of Dr. Paul A. Younge.

CASE 14. K. T. (M.G.H. 130139), a 57-year-old woman, complained of an increase in vaginal discharge of 3 months' duration. There had been no blood. The cervix appeared normal to inspection. A vaginal smear on February 24, 1947, was positive. Biopsy on March 21 disclosed epidermoid carcinoma in situ.

CASE 15. H. V. (private case), a 49-year-old woman with no symptoms, after 3 months of treatment with estrogens for arthralgia noted 1 day of spotting. A vaginal smear on November 27, 1946, was positive. Biopsy on December 16 revealed chronic cervicitis. The pathological diagnosis was carcinoma in situ.

CASE 16. P. F. (M.G.H. 545101), a 42-year-old woman, was admitted for a mass in the breast that proved to be due to chronic cystic mastitis. There were no pelvic symptoms. Examination showed an area of leukoplakia on the cervix. Vaginal smears on December 10, 12 and 21, 1946, were positive. Biopsy on December 14 disclosed leukoplakia. The pathological diagnosis on the excised uterus was cervical carcinoma in situ.

CASE 17. D. T. (private case), a 31-year-old woman, had had continuous flowing for 4 months. There was a rough area on the left lip. A vaginal smear was negative. Biopsy revealed chronic cervicitis. The pathological diagnosis was cervical carcinoma in situ.

SUMMARY AND CONCLUSIONS

Twelve cases of early cervical carcinoma are reported in which the vaginal smears were positive for tumor cells in 10. Two of these patients had had no symptoms referable to the lesion. In 9 cases in which biopsy was performed, a diagnosis of cancer on the initial specimen was made in only 3.

Preinvasive carcinoma exists for several years before clinical evidence of the disease appears. When a carcinomatous lesion of the cervix manifests itself by symptoms it is often no longer in the early stage. Early diagnosis is essential if cancer of the cervix is to be cured. Diagnosis of preinvasive carcinoma of the cervix is possible by study of the vaginal smear.

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AN OUTBREAK OF SMALLPOX IN AMERICAN MILITARY PERSONNEL IN JAPAN

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PHILADELPHIA

SMALLPOX, an ancient disease whose prevention has been so readily achieved since the days of Jenner, continues to manifest itself. Even in some parts of the United States the record is shameful. A number of states, especially in the West and Northwest, do not have compulsory vaccination laws. In 1924 Minneapolis had 860 cases, with 219 deaths. In 1931 a total of 30,232 cases were reported from the country as a whole. More recently, however, the morbidity figures have markedly declined, there having been reported only 346 cases in this country in 1945.

Most physicians have never seen a case of smallpox, particularly those who practice in states in which vaccination laws are rigidly enforced. Furthermore, the majority of cases that have been described in this country in recent years have been relatively mild. On the other hand, North Africa and the Far East, especially India, are hotbeds of hemorrhagic smallpox.

Unquestionably, the health of the armed forces during the recent conflict was the best ever on record. The program of preventive medicine as outlined and carried out was generally quite adequate. Consequently, an outbreak of smallpox among American troops stationed in Japan came as a surprise and as a rude jolt.

Smallpox, an acute infectious disease of virus etiology, is highly contagious and is transmitted from man to man and by fomites. Standard textbooks present a full discussion of the etiology, incidence, pathology and diagnosis. The case reports presented below, however, will serve to clarify, illustrate and amplify virtually all aspects of the disease.

During January, 1946, a total of 17 patients with smallpox were admitted to a station hospital. I was ordered to this institution at the time of the peak of the outbreak to lend assistance. Since this afforded a rare opportunity, a detailed report of these cases is considered justifiable.

CASE REPORTS

CASE 1. This man was on duty in December, 1945, in a Japanese town. In his duties, which consisted of guarding and inspecting a Korean repatriation area, he came in contact with countless natives. Several cases of smallpox among civilians had recently occurred. On December 17 the patient was admitted to a medical battalion clearing station because of complaints later shown to have been the prodrome of smallpox. On December 19 a rash broke out. Several days later the diagnosis of smallpox was made. The course was stormy, but the patient began to improve, and on January 12, 1946, he was transferred to a station hospital for convalescence. At that time, he was almost entirely free from crusts. The patient stated that he had been vaccinated in childhood, but he recalled no definite "take," nor did he remember having had a scar on either arm. A vaccination in August, 1944, while he was in the Army, did not appear to have been successful.

CASE 2. This man was in the same ward in the medical battalion clearing station as the patient in Case 1. He had a cellulitis of the left ankle at the time of that patient's admission. On December 29 he was vaccinated. On the next day he began to develop symptoms that eventually showed themselves to have been the prodrome of smallpox. On January 2 he was transferred to the station hospital without a definite diagnosis. A reaction to the vaccination was suspected. The patient, however, was isolated on the fourth floor of the hospital. Medical aseptic technic was said to have been employed. On January 6 the diagnosis of smallpox was made. Two days later the patient expired of hemorrhagic smallpox. The vaccination on entrance into the Army was reported to have been "immune." A childhood vaccination had not been successful.

CASE 3. This man was one of a series of patient contacts in the station hospital. (There was no history of any direct contact with the patient in Case 1, who had been isolated, but epidemiologically speaking there is no other plausible explanation for these cases.) He was admitted to the station hospital on December 2 with the diagnosis of infectious hepatitis. He was placed in the hepatitis ward on the fourth floor. On January 14 he developed symptoms that were later shown to have been the prodrome of smallpox. Three days later a rash appeared. The patient was isolated. He ran a typical course of severe pustular smallpox, from which he convalesced slowly. He recalled a childhood vaccination but could not remember whether it had been successful. In July, 1944, a vaccination done in the Army had apparently been unsuccessful.

CASE 4. On December 30 this patient was admitted to the third floor of the station hospital with the diagnosis of malaria. He received routine antimalarial therapy, and on January 8 he was returned to duty. Six days later he developed prodromal symptoms of smallpox. On January 16 he was readmitted to the hospital. He was again placed on the third floor. He developed a rash on January 18 and expired from hemorrhagic smallpox on January 23. A vaccination on November 28, 1944, had been reported as "immune." A childhood vaccination at the age of 7 years was said to have been successful.

Autopsy. Post-mortem examination disclosed the shotty, hemorrhagic, umbilicated vesicles covering most of body, confluent on the face, chest, abdomen and proximal portion of the extremities. The lungs showed congestion, edema and hemorrhage in all lobes. There was acute, hemorrhagic tracheobronchitis. Petechial hemorrhages over all the pleural surfaces were observed. Petechiae were scattered over the pericardium and endocardium. There was mild endocarditis. The gastrointestinal tract showed petechial hemorrhages and superficial erosions of the esophagus, stomach and the small and large intestines. A moderate hepatomegaly, with diffuse fatty changes in the middle and central zones, was observed. The genitourinary system revealed chronic interstitial nephritis, bilateral, with massive hemorrhage, hemorrhage into the renal pelves, congestion and cloudy swelling. There was bilateral ureteral congestion and hemorrhage, as well as blood clots in both ureters and in the bladder. There were petechial hemorrhages of the bladder mucosa. Acute orchitis with interstitial congestion and hemorrhage was noted. Petechial hemorrhages were observed over the tunica vaginalis. There was a moderate splenomegaly.

CASE 5. On January 4 this man was admitted to the third floor of the station hospital with a diagnosis of malaria.

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He was returned to duty on January 13. Four days later he developed symptoms that were later shown to have been the prodrome of smallpox. On January 21 he was readmitted to the third floor. On the same day a flush of the skin was seen, and he was transferred to the isolation ward on the fourth floor. The patient developed a typical case of hemorrhagic smallpox and expired on January 26. There had been no vaccination in childhood. A vaccination on November 28 was reported to have been "immune."

CASE 6. On November 19 this man was admitted to the fourth floor of the station hospital for the study of a chronic gastrointestinal complaint. Eventually, diagnoses of hookworm infection, bronchitis and sinusitis were made. He was returned to duty on January 2. On the next day, he was readmitted because of the persistence of some of the symptoms. He was again hospitalized on the fourth floor. On January 17 he developed symptoms that were later shown to have been the prodrome of smallpox. On January 18 he was somewhat flushed, and on the following day the onset of a rash caused him to be admitted to an isolation ward. He developed hemorrhagic smallpox and expired on January 26. Information regarding a childhood vaccination was not available. A vaccination on January 9, 1945, was reported to have been "immune."

CASE 7. This man was admitted to the third floor of the station hospital with the diagnosis of malaria. This diagnosis was sustained, and on January 14 he developed symptoms that were later shown to have been the prodrome of smallpox. He was readmitted on January 21 and isolated in the communicable-disease section. A flush was already visible. He developed a typical case of hemorrhagic smallpox and expired on January 26. No history regarding a smallpox vaccination in childhood was obtainable. A vaccination on March 20, 1944, was reported to have been "immune."

CASE 8. This patient gave a history of having frequently visited — in the early weeks of January — a patient on the fourth floor who was said to have been convalescing from scarlet fever. The contact with this floor is a possible clue, although it must be admitted that other unknown contacts may have given this man his infection. He developed on January 22 a typical case of hemorrhagic smallpox and expired on January 28. He had not been vaccinated on January 19 because he had been on duty away from his unit at that time.

CASE 9. It is difficult to place this man's contact with the patients in the other cases. It might be speculated that, during the prodromal period of the disease in Case 4, there had been contact between these men at the Red Cross building, where many of the men gathered. The incubation period would have had to be short to fit into this scheme — although such a contact was possible, it is admitted that it represents speculation and cannot be a certain deduction. No definite history of childhood vaccination was obtainable. A vaccination on November 17, 1944, was said to have been "immune." This patient developed the prodrome of smallpox on January 17. On January 21 he was admitted directly to the communicable-disease section on the fourth floor and isolated. A rash was first seen on January 22. He ran a typical course for hemorrhagic smallpox and expired five days later.

CASE 10. This man was a member of the same regiment as the patients in Cases 6 and 9, and a similar speculation regarding his contacts is necessary. On January 17 he developed what was later shown to have been the prodrome of smallpox. On January 19 he was admitted to the hospital. A rash developed on the following day, and he was isolated. He developed a typical case of hemorrhagic smallpox and expired on January 28. A vaccination in November, 1944, was reported to have been "vaccinoid" (accelerated). The patient had never been vaccinated in childhood.

CASE 11. No direct contact could be ascertained in this case. The patient was admitted to the station hospital on January 23 by transfer from the medical battalion clearing station, where he had been admitted several days previously. The diagnosis was smallpox. This patient developed a typical case of hemorrhagic smallpox and expired on January 25. He had never been vaccinated in childhood. A record of his vaccinations in the Army could not be obtained.

CASE 12. This was the first of a series of cases among hospital personnel. In these cases, it seems quite likely that the patient in Case 1 was the source of the contagion. Although it was impossible to determine any contact, this demonstrates the high degree of contagion in smallpox. This patient was on duty in the detachment office and also had duties in the information and education section. Thus, he had numerous contacts among the patients. On January 15 he complained of symptoms later shown to have been the prodrome of smallpox. On the following day he was admitted to the fourth floor of the station hospital. He was not strictly isolated at first. On January 21 a rash appeared. He was strictly isolated from then on. The patient developed a severe pustular type of smallpox and subsequently showed hemorrhagic complications. He expired on January 26. An "immune" reaction to vaccination on March 13, 1945, was recorded. Vaccination was repeated on January 19. No reading was possible because of the rash. No definite history of childhood vaccination was obtained.

CASE 13. This man had numerous duties in the hospital. During the time of probable infection, however, his chief point of contact was attendance at the Red Cross canteen in the hospital. This was a popular place and served as a social center for the entire hospital. On January 17 the patient developed symptoms later shown to have been the prodrome of smallpox. He was admitted to the hospital on January 19. A rash was visible at that time, and he was immediately placed in isolation on the fourth floor. This patient developed a typical case of pustular smallpox and recovered. Interrogation obtained no clear history of childhood vaccination. A vaccination in June, 1945, had produced a questionable reaction.

CASE 14. This man had many duties in the hospital laundry that brought him into intimate contact with the dirty linen. It should be emphasized that the contaminated linens were always autoclaved before being sent to the laundry. It is quite possible, however, that by mistake a contaminated sheet was sent directly to the laundry. This seems to have been the source of the infection in this case. On January 16 symptoms that were later shown to have been the prodrome of smallpox developed. The patient was admitted to the hospital on January 19 and was put on the fourth floor. On the following day he developed a rash and was promptly isolated. He developed a typical case of pustular smallpox and recovered completely. There was no history of vaccination in childhood. A vaccination on December 18, 1943, was reported to have been "immune."

CASE 15. This is one of 3 extremely mild cases. There was no doubt about the diagnosis, however. This patient was the hospital carpenter and utility man and had occasion to be in many parts of the hospital. On January 18 he felt "grippy." On the same day he was admitted to the fourth floor of the hospital. On January 21 he felt better, and simultaneously an eruption came out, first on the face. He ran an uneventful course and recovered completely. A vaccination on February 9, 1943, was reported as "vaccinoid."

CASE 16. This man was a surgical technician working on the fifth floor of the station hospital. He had no direct contact with any of the cases on the fourth floor, but he was about the hospital to some extent. On January 18 he was admitted to the fourth floor of the hospital with symptoms characteristic of the prodrome of smallpox; these had begun on January 14. The fever dropped on January 21, and on the following day he developed symptoms of a mild case of smallpox, showing a rash predominantly on the face. There was no definite history of a childhood vaccination. A vaccination on November 16, 1943, was reported to have been "vaccinoid." A vaccination on January 20, 1946, was doubtful in reaction.

CASE 17. This Red Cross worker ran the canteen and was exposed to many people. On January 19 she developed symptoms later shown to have been the prodrome of smallpox. She was admitted to the hospital on the same day. On January 23 the rash came out. This patient ran a mild course of smallpox. There was a history of a successful childhood vaccination. Vaccinations in September, 1944, and February, 1945, were reported to have been doubtful.

Laboratory Studies

In the mild cases and in the typical, pustular cases there were, generally speaking, no outstanding changes in the red-cell counts and in the hemoglobin concentrations. In the hemorrhagic cases, however, there was a distinct drop in the red-cell counts and in the hemoglobin concentrations, with one exception the declines varying from 5 to 15 per cent. One case showed a terminal drop from an original red-cell count of 4,500,000 to 1,900,000. The hemoglobin fell accordingly. The administration of daily small blood transfusions undoubtedly tended to minimize the anemia.

Most cases showed some leukocytosis. In the mild cases this was not marked. In the pustular cases the white-cell counts went up during the pustular stage, averaging 12,850 to 15,700. The hemorrhagic cases showed consistently a greater leukocytosis than the others, with counts of 14,000 to 23,000, and this was maintained during the entire illness in most cases. One of the mild, pustular cases developed a white-cell count of 4560 at the beginning. The differential count was chiefly remarkable because of a moderately pronounced lymphocytosis. A typical differential count was 56 per cent neutrophils and 44 per cent lymphocytes. The platelet counts, which were determined in 4 hemorrhagic cases, were entirely within normal limits, the figures varying between 200,000 and 240,000. The bleeding time was increased, averaging about 12 minutes. This could also be verified by the prolonged bleeding seen at the sites of injections and venipuncture. The coagulation time was observed in only 1 case, in which it was 14 minutes by the capillary method.

The mild cases, and the typical, pustular cases showed no remarkable urinary findings. In the hemorrhagic cases, the daily urinary output was satisfactory — averaging 1500 cc. — until the last two or three days of illness. Albumin and coarsely and finely granular casts were occasionally found in the sediments, and gross hematuria usually occurred two or three days before death. In 7 fatal hemorrhagic cases the patients were anuric during the last day of life, but in the other 3, the hematuria appeared to subside just prior to death.

Clinical Course

The clinical course in these cases suggested that the prodrome gave no indication of the type of smallpox to be anticipated. The symptoms characterizing the prodromal stages run three or four days. Fever, headache, backache, general malaise, anorexia, chilly sensation, nausea and vomiting and sore throat make up the picture. As a whole, the clinician is likely to interpret this symptom complex as an influenza-like entity.

All patients in this outbreak were in the third decade of life.

Mild Cases

In the mild cases a definite fall in temperature set in after the three-day period, along with a return of a feeling of well-being. Suddenly a maculopapular rash was noticed, usually appearing first on the face and upper extremities. The rash revealed a somewhat crop-like tendency. There was no notable secondary rise in temperature.

The lesions were not so firm and deep seated as those in the severer cases. The eruption, however, was preponderantly on the face and extremities. After thirty-six to forty-eight hours the vesicular stage was reached, and twenty-four hours later, the vesicles took on a yellowish hue, becoming pustular. Some lesions were seen on the palms of the hands; others showed umbilication, and a few revealed a late, secondary hemorrhage into the lesion. Crust formation was seen about five to seven days after the onset of the rash. A few lesions did not show a distinct pustular stage. The crusts fell off about two weeks after the onset of the rash. No notable pockmarking was seen.

These patients never appeared extremely ill. It is questionable whether, without the presence of the severe cases, a proper diagnosis would have been made. There was no doubt of the diagnosis, however, under the circumstances.

In the mild cases 2 patients gave fairly definite histories of successful childhood vaccination. In the other case, no definite information could be obtained; a typical scar was not found.

Pustular Cases

The cases in the typical pustular group were consistent with the usual textbook picture. With the onset of the rash there was a drop in temperature for about twenty-four hours. The temperature then ran an irregular septic course, ranging between 99 and 103°F. for about fourteen days. The evolution of the rash was from macule to papule to vesicle to pustule to crust. In the papular stage the case was reminiscent of a severe measles rash. There were considerable catarrhal symptoms, including lacrimation, rhinitis and cough and generalized cervical adenopathy, as well as some axillary adenopathy. During the pustular stage the patients were quite toxic. The falling off of the crusts was a large-scale process, with the bedclothing and floor littered with coarse, brawny scales. The scales and crusts did not disappear entirely until about six weeks from the onset of the illness. Considerable alopecia occurred, and pockmarking was extensive.

Hemorrhagic Cases

Variola pustulosa hemorrhagica. Two patients had an onset similar to that in the other pustular cases. During the vesicular stage, when the disease appeared to be running a fairly typical course, subconjunctival hemorrhages were seen, and hemorrhages into most of the vesicles appeared. The cough

became worse, and blood-streaked sputum was seen. Numerous small gray ulcerations were observed in the tonsillar fossa at that time. Hematuria set in but was of a lesser degree than that in the other hemorrhagic cases. The cough became worse, the condition became critical, and the patient expired rather suddenly. There was no characteristic fall in temperature at the time of the onset of the rash.

Purpura variolosa. Eight of the fatal cases were of the purpura-variolosa type. After the prodromal period, these patients uniformly presented a scarlatiniform flushing of the face, neck and chest. The extremities showed a mottled, reddish appearance. Subconjunctival hemorrhages were seen early. About thirty-six to forty-eight hours later the patients exhibited a purpuric eruption that was more or less generalized, not marked on the face but especially prominent on the chest, forearms, feet and legs. At about that time the face and extremities (especially the arms) became edematous, the eyelids swelled, and the conjunctivas were markedly hemorrhagic. Large quantities of thick mucus streaked with blood were raised; cough was marked, and efforts to swallow were frequently unsuccessful. Drooling occurred. The voice was thick and weak. Enemas yielded returns of bloody water. The sites of injections bled for minutes to hours. After several days vesicles formed, especially on the chest, with a few on the extremities. Some of the vesicles were hemorrhagic.

Three patients took on a slate-gray appearance. Four had small, closely spaced vesicles over the chest and extremities.

All fatal cases exhibited coarse, moist rales and rhonchi in the chest. The patients became extremely toxic. Large blebs on the wrists and ankles were noted prior to exitus in 4 cases. Two patients never developed much of an eruption, except for the slate-gray hemorrhagic appearance.

As the exitus approached, the patients became extremely stuporous. Respirations were somewhat labored, and coarse, moist rales were audible even without the use of a stethoscope. The impression was obtained that the patients were overwhelmed by the infection.

Therapy

Hemorrhagic Cases

It is at once apparent that there is still no satisfactory therapy for the hemorrhagic cases. The mortality is virtually 100 per cent. In this series there were no recoveries. All the patients in this group received penicillin therapy. At first, the dose was 20,000 units intramuscularly every three hours. Later, this was increased to 40,000 units a dose. There was no tangible effect from the penicillin therapy. Daily blood transfusions were given in amounts of 150 cc. daily. The fluid intake was largely handled by the intravenous route, since the oral intake in these cases was greatly restricted owing to difficulty in swallowing. Each day, 2000 cc. of 5 per cent glucose in physiologic saline solution or 10 per cent glucose in distilled water was given. Plasma was administered in amounts of 250 cc. daily.

Adrenocortical extract was also given to 5 patients. No visible benefits were seen. Vitamins K and C were also administered, the intravenous route being utilized for the most part. The first patient to convalesce was employed as a donor for the other serious cases. Ten cubic centimeters of his whole blood was given twice to 5 of the seriously ill patients. This amount was no doubt inadequate.

Locally, various preparations were used. Petrolatum, sodium bicarbonate and plain calamine lotion were tried. It was difficult to furnish relief. All the patients complained that the light hurt their eyes, and a darkened room afforded them much relief. A 10 per cent solution of mild silver proteinate was instilled into the eyes several times a day.

Aspiration, oral hygiene, sedation and oxygen therapy were employed on indication. It is obvious that none of these measures were helpful in preventing the final outcome of the disease. In these cases, despite modern science, one stands by helplessly at the sick bed. The situation is characteristic of that in many virus diseases: once the cells have been invaded, therapy is generally of no avail.

Pustular Cases

In these cases the advent of penicillin represents a therapeutic advance. Although it is difficult to draw definite conclusions from the cases presented above, the employment of penicillin is certainly logical. The pustular stage should indeed be favorably influenced by this antibiotic. Blood transfusions and parenteral therapy were given when needed, as were local applications and other palliative and supportive measures.

Mild Cases

The treatment in these cases was minimal. One patient received penicillin therapy for three days.

Discussion

In the study of these cases there were a number of interesting factors for consideration. Unfortunately, a post-mortem examination was possible in only 1 case. Facilities for autopsy were poor, and in view of the highly contagious nature of the disease, it was considered prudent to minimize exposure. It must be pointed out that at the time of the outbreak the situation about the institution was one of some understandable uneasiness. The wide variety of the contacts instilled a wholesome awe for the ease with which the disease could be spread and even some doubt concerning the efficacy of vaccination. Fortunately, the value of smallpox vaccination was again demonstrated beyond doubt.

Many of the unvaccinated patients had come from states where compulsory vaccination laws exist. In the 3 mild cases clear-cut histories of childhood

vaccination were obtained. Two patients still showed a scar. A vaccinoid reaction within a three-year period prior to the attack was recorded in 2 cases.

In the severe, pustular cases, the history revealed either an unsuccessful childhood vaccination or none at all. Vaccinations done within a three-year period were recorded as "immune" reactions in 3 cases. In another case no record was available.

In only 1 of the fatal cases was there a fairly reliable history of a successful childhood vaccination. Four patients apparently had received no childhood vaccination, and the remaining 5 provided an unreliable history. The severe eruption made identification of any old scars impossible. In 8 of the fatal cases the patient had had an "immune" reaction within three years of the illness. In 1 case the reaction was reported as "vaccinoid," and in another the immunization register was unobtainable.

Despite the incompleteness of some of the vaccination data, however, certain points stand out. Vaccination is not so universal in the United States as one might think. Even in states with compulsory vaccination laws there seem to be many slips. It seems to be fairly well established that vaccination in the armed forces has not always been performed with sufficient care — especially regarding the recording of reactions. Interrogation of some of the patients caused doubt whether they had ever received successful vaccinations in their lives. There had at times been a tendency to fail to read and to interpret the vaccination properly. It seems quite certain that many "immune reactions" should have been recorded as "no take."

In addition, it is apparent that even successful vaccination does not necessarily confer lifelong immunity. A successful childhood vaccination, however, frequently prevents the attack from becoming hemorrhagic. Whenever an area in which cases of smallpox have occurred is entered, all persons should be vaccinated without delay.

The universal revaccination of all troops and personnel, the enforcement of a working quarantine on the hospital and the strict isolation of all cases, contacts and suspects quickly terminated this outbreak.

The high degree of contagion of smallpox is again demonstrated. All but 3 of the cases were definitely related to Case 1. This fact, however, indicates that the contact need only be fleeting. Presumably, the effectiveness of the isolation technic used at the station hospital was average, although breaks obviously occurred. On the other hand, there seems to be no reason to assume that smallpox is spread in a mysterious way by winds blowing from "pest houses" and the like. Medical aseptic technic employed by an adequately vaccinated personnel, with the patient completely isolated, should afford adequate protection. It is gratifying and reassuring to note that none of the physicians, nurses or attendants in intimate contact with these patients contracted the disease. Such persons were promptly revaccinated.

Until the fact of an epidemic has been established, the diagnosis of a case early in the disease is frequently difficult. This applies especially to the hemorrhagic cases, with their early scarlatiniform flush. Many of the cases also resembled measles. The mild cases naturally bring up the question of chicken pox. The long prodromal period is of help in suggesting the possibility of smallpox.

Apparently, hemorrhagic smallpox is merely a severe form of the disease. There seems to be no validity in postulating a hemorrhagic strain of the virus, since all varieties of smallpox originated from the same source. It is probable that the degree of exposure and the extent of the immunity had a bearing on this matter.

The incubation period in these cases averaged about two weeks, which is in harmony with the usual experience. The time can be less than that period, however, and also of greater duration.

Summary

An outbreak of 17 cases of smallpox, with 10 deaths, is reported and discussed.

Hemorrhagic cases are highly fatal, representing a 100 per cent mortality in this series.

Penicillin therapy is of promise in cases of the pustular variety.

The high degree of contagion of smallpox is again demonstrated.

The difficulty of early diagnosis is emphasized, especially among the first cases of an outbreak.

Vaccination is still the only satisfactory answer to the problem. A scrutiny of the vaccination history in these cases showed that the protection had been inadequate for the most part.

In the face of actual cases, the entire personnel must be revaccinated.

Fresh vaccine, proper vaccinating technic and the correct interpretation of the reactions are essential to an effective program of prevention.

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HEMOGLOBINEMIA ACCOMPANYING TRANSURETHRAL RESECTION OF THE PROSTATE*

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THE removal of the prostate by the transurethral method, together with newer methods of preoperative and postoperative care of the patient by the use of fluid therapy and antibiotics, has increased the number of patients who may safely be subjected to prostatectomy. In spite of the present refinements, transurethral resection carries an appreciable — although low — mortality. One of the disturbing complications sometimes noted is a temporary oliguria or even anuria. A fatal case of renal failure following transurethral prostatectomy called attention to the possibility that intravascular hemolysis played a part in its causation. The following is a brief abstract of this case:

A 62-year-old salesman was admitted to the hospital on July 5, 1945, because of inability to urinate of 12 hours' duration. He gave a history of increasing prostatism during the preceding year.

Physical examination was negative except for a blood pressure of 200/110 and a markedly distended bladder. The prostate was enlarged about twice the normal size on rectal examination, but showed no evidence of cancer.

Examination of the blood revealed a hemoglobin of 9 gm. per 100 cc. and a white-cell count of 9800, with a normal differential count. The urine, which showed a specific gravity that ranged between 1.006 and 1.012, gave a +++ test for protein; the sediment contained an occasional red and white cell. The blood urea nitrogen was 8 mg. per 100 cc., and the phenolsulfonephthalein test showed 39 per cent excretion of the dye in 30 minutes and 65 per cent at the end of 2 hours.

After catheterization the patient was able to void spontaneously, and on the 4th hospital day a transurethral prostatectomy was performed under combined spinal and Pentothal anesthesia. A moderate amount of bleeding occurred during the resection, and a transfusion of 500 cc. of citrated blood, found on crossmatching to be compatible, was given while the operation was in progress. This was followed after operation by 1000 cc. of distilled water containing 5 per cent dextrose, and later a second transfusion of 500 cc. of citrated blood was given because of continued bleeding and a low blood pressure. A sample of serum obtained before the second transfusion, for the purpose of crossmatching, was observed to show marked hemolysis.

On the 1st postoperative day the patient appeared ill; he was restless and nauseated, and the urinary output was low. Bleeding was slight. The blood urea nitrogen was 65 mg. per 100 cc. In the afternoon the blood serum showed a hemochromogen level§ of 199 mg. and a total bilirubin of 2.08 mg. per 100 cc. On the 2nd day the hemochromogen level was 130 mg. and total bilirubin 0.77 mg. per 100 cc., and 3 days later the hemochromogen level was 33 mg. per 100 cc.

Throughout the 1st postoperative week the patient produced only a small amount of urine, the total output being about 1500 cc. The blood urea nitrogen rose to 90 mg. per 100 cc. on the 3rd and to 120 mg. on the 5th postoperative day. During that time the patient received supportive therapy, which included intravenous fluids, whole blood, plasma and amino acids, since he was unable to take anything by mouth. On the 8th day a diuresis of 1400 cc. occurred. Thereafter, the patient maintained an output of over 2000 cc. of urine daily, but the specific gravity was persistently low, and the urea concentration of the urine varied between only 121 mg. and 285 mg. per 100 cc. (normal 2500 to 3000 mg.). This, with blood urea nitrogen levels between 110 and 140 mg. per 100 cc., demonstrated an almost complete lack of concentrating power on the part of the kidneys. On the 11th postoperative day the patient developed auricular fibrillation, dying on the next day of cardiac failure. Permission for autopsy was not obtained.

It seemed evident that in this case intravascular hemolysis, as demonstrated by an elevated serum hemochromogen level, was followed by hemoglobinuric nephrosis. An intrinsic hemolytic anemia seemed an unlikely cause for this reaction, since all laboratory tests, including cold hemolysis, acid hemolysis, hypotonic fragility, sickling preparation and cold agglutination, were within normal limits. The two transfusions of bank blood that the patient received had been rechecked for type and compatibility, and both were found to be compatible. One was Type O and Rh positive, and the other Type O and Rh negative; the patient was Type O and Rh positive. The small dose of sulfadiazine — 1 gm. daily for four days — given preoperatively in the absence of other evidence of sensitivity, spherocytosis or abnormal agglutination did not account for the severe hemolytic reaction.

It was suggested that the operation of transurethral prostatectomy in itself was responsible for the hemolysis.|| Accordingly, the blood of 15 consecutive patients undergoing transurethral resection was tested for the presence of hemolysis¶ by measurement of the serum hemochromogen level before and immediately at the close of the procedure (Table 1). All patients were operated on by the technic described by Nesbit.[2] A Bovie generator was used, and distilled water for irrigation was obtained from a reservoir about 75 cm. above the level of the bladder.

In this group 10 patients (66 per cent) showed significant hemolysis, the blood hemochromogen levels after operation ranging between 24 and 316 mg. per 100 cc. In 3 cases the level was over 200 mg. per 100 cc. — that is, within the range observed in the patient with renal failure described above.

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§The blood hemochromogen level[1] is a measure of all pyrol-iron pigments free in the blood stream, including not only hemoglobin but also methemoglobin, hematin and methemalbumin. The principal substance found free in the blood stream immediately after hemolysis has taken place is hemoglobin, but if determinations are made a number of hours later, appreciable amounts of the other substances may also be found. These other substances are derived from the free hemoglobin originally present. The hemochromogen levels mentioned below are therefore a measure of the amount of hemolysis that had taken place.

||This suggestion was made by Dr. Joseph Ross.

¶A sample of blood was drawn from an antecubital vein, allowed to clot and centrifuged, and the supernatant serum separated within thirty minutes. All transfers of blood were made under mineral oil. Hemochromogen levels were determined by the method of Flink and Watson.[1]

All but one of the patients showing hemolysis made a good recovery and gave no evidence during this period of any renal damage or increased nitrogen retention as a result of the operation with its attendant hemolysis. One patient, however, with a hemochromogen level of 220 mg. per 100 cc., had temporary oliguria on the day after operation, despite an adequate fluid intake (3200 cc.). This lasted for only about twelve hours, during which almost no urine was formed, and was followed by a diuresis lasting for two days with an output of over 2000 cc. on each day. The damage to the kidneys was reflected by a postoperative rise in the blood urea nitrogen to 53 mg. per 100 cc. on the second day, which returned slowly to normal after ten days.

At least two possibilities are suggested as the cause of the observed hemolysis: local action of the electric energy of the instrument on blood-containing tissues, and the penetration of the irrigating fluid (distilled water) into the blood stream either by lymphatic absorption or into venous channels opened by operation, with resulting hemolysis.

The first possibility seemed remote, in view of the absence of hemolysis in patients subjected to fulguration of bladder papillomas by the same type of electric current as that used in the resectoscope.

To investigate the possibility that distilled water used for irrigation during the resection gained access to the blood stream, sodium salicylate, in amounts of 1 gm. per liter, was added. This substance was chosen to label the irrigating fluid, since a method of accurately determining its presence in small amounts in the blood has been described by Brodie et al.[3] Also, it was found that in this concentration in the irrigating fluid, sodium salicylate caused no interference with the cutting action of the resectoscope. The blood levels of sodium salicylate to be expected an hour after the administration of varying amounts of this drug were investigated in several patients prior to its use in transurethral prostatectomy (Fig. 1).

TABLE 1. *Hemochromogen Levels before and after Transurethral Resection of the Prostate.*

PATIENT	AGE	DIAGNOSIS	DURATION OF OPERATION	TISSUE REMOVED	TRANSFUSION*	SERUM HEMOCHROMOGEN LEVEL	
						BEFORE OPERATION	AFTER OPERATION
	yr.		*min.*	*gm.*	*cc.*	*mg./100 cc.*	*mg./100 cc.*
W. A.	65	Benign prostatic hypertrophy	50	10	—	4	11
C. T.	42	Benign prostatic hypertrophy	75	—	300	1	51
S. W.	59	Benign prostatic hypertrophy	60	29	—	—	52
B. B.	66	Benign prostatic hypertrophy	40	4	—	—	13
H. H.	72	Carcinoma of prostate	35	2	—	—	52
G. S.	63	Benign prostatic hypertrophy	60	12	—	—	265
C. F.	68	Carcinoma of prostate	35	16	—	2	2
C. G.	68	Benign prostatic hypertrophy	35	6	—	4	32
R. M.	59	Benign prostatic hypertrophy	120	18	—	—	24
J. R.	73	Benign prostatic hypertrophy	75	10	—	7	110
J. K.†	65	Benign prostatic hypertrophy	55	19	500	—	220
J. P.	71	Benign prostatic hypertrophy	50	10	—	—	65
S. McP.	77	Carcinoma of prostate	60	16	—	4	6
J. B.	64	Carcinoma of prostate	70	13	500	—	316
W. L.	70	Benign prostatic hypertrophy	85	—	—	8	12

*Blood given after hemochromogen determination.
†Patient had oliguria for one day.

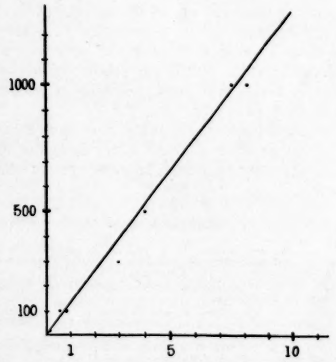

FIGURE 1. *Sodium Salicylate Blood Levels following the Intravenous Injection of the Drug.*

The ordinate represents the amount of drug injected (in milligrams), and the abscissa, the blood level (in milligrams per 100 cc.) one hour after injection.

TABLE 2. *Hemolytic Effect of Water Administered Intravenously in Man.*

AMOUNT OF WATER INJECTED	SERUM HEMOCHROMOGEN LEVEL AFTER INJECTION
cc.	*mg./100 cc.*
100	11
100	21
200	45
300	47
600	201*
900	380*

*Hemoglobinuria present.

Three patients were subjected to transurethral prostatectomy, an irrigating fluid containing 1 gm. of sodium salicylate in 1000 cc. of distilled water being used. In 2 cases no sodium salicylate could be demonstrated in the blood, nor was there any hemolysis. The third patient, in whom a large amount of hemolysis occurred, — a hemochromogen level of 316 mg. per 100 cc., — also showed the presence of sodium salicylate, the blood hemochromogen level at the end of operation being 7.5 mg. per 100 cc. This level of sodium salicylate is to be expected an hour after the intravenous administration of about 1 gm. of this substance and therefore represents the entrance of about 1000 cc. of irrigating solution into the blood stream.

In the patients studied it has been assumed that the distilled water entering the circulation caused hemolysis of the blood. Such a fluid is well known to be hemolytic for red cells in vitro. Krumbhaar,[4] in experiments on dogs, found that the intravenous injection of distilled water of 2 to 3 per cent of the body weight produced hemoglobinemia and hemoglobinuria, and that the speed of injection — from three to forty-five minutes — had little effect on the amount of hemolysis. To check the validity of these data for man, distilled water was given intravenously in amounts from 100 to 900 cc. to several patients. The smaller amounts were given rapidly over one or two minutes. The larger amounts (300 to 900 cc.) were given slowly over a period of one to four hours. Appreciable hemolysis was produced in every case (Table 2). In no case was there any reaction or evidence of renal dysfunction after injection. The data suggest that amounts of distilled water in the neighborhood of 500 cc. or more are necessary to produce hemoglobinuria.

Discussion

Intravascular hemolysis is a frequent consequence of transurethral prostatectomy. Evidence has been presented that distilled water used for irrigation gains access to the blood stream and that it is capable of producing the degree of hemolysis observed. The demonstration by Nesbit of free venous bleeding from veins of the prostatic capsule injured during operation makes it quite evident that these are a portal of entry for the fluid, when its pressure exceeds that in the veins. Although the fluid used for irrigation already contains hemolyzed blood, this accounts for only a fraction of the hemolysis observed.

There is no reason to believe that the renal damage resulting from hemolysis during transurethral resection differs essentially from other types of hemoglobinuric nephrosis. The pathogenesis of the renal failure in these conditions is not yet well understood, as pointed out by Bradley.[5] Hemoglobinemia is not the sole cause, for comparable hemolysis has been produced[6] and comparable amounts of distilled water have been injected intravenously[7] without causing renal damage. Probably, blood loss and renal ischemia[8, 9] both of which may well occur during transurethral resection, play an important contributory role in the pathogenesis of the renal lesion.

Regarding the prevention of such hemolysis, in view of the possibility that fluid enters the circulation through venous channels opened during operation, the use of irrigating fluid under as low a pressure as possible and in as small a quantity as is consistent with clear vision is self-evident. The employment of a fluid such as physiologic saline solution, which is well borne in the general blood stream, is precluded because the cutting current of the resectoscope cannot be efficiently produced in a medium containing strong electrolytes. The addition of some nonelectrolyte, such as glucose, to the irrigating fluid has been tried but has not been found entirely satisfactory because of impaired vision.

Summary

A fatal case of renal failure following transurethral prostatectomy is described. Death was caused by massive intravascular hemolysis occurring during the operation.

Fifteen patients undergoing transurethral prostatectomy were studied for the presence of hemolysis, by measurement of the serum hemochromogen level immediately at the termination of operation. In 10 cases a significant amount of hemolysis could be demonstrated, with extremely high hemochromogen levels in 3 cases, resulting in temporary oliguria in 1 patient.

Evidence is presented that this hemolysis is caused by the entrance of distilled water into the blood stream through venous channels opened during operation.

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MEDICAL PROGRESS

INDUSTRIAL MEDICINE

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INDUSTRIAL medicine in the first postwar year continued to show the influence of the lessons learned from the war. New ideas, new methods, new procedures and new viewpoints that originated in war experiences were encountered everywhere. The progress made may be divided into two groups, technical and social.

Technical advances were manifold and should prove of enduring value. General safety programs in many war industries reached a new high level, with gratifying results. In the munitions industry lessons learned from World War I were remembered and utilized; new experiences were added, and safety devices introduced that proved highly effective. As a result fatalities among munition workers during the war were remarkably small compared with the records of 1918. Industrial hygiene also profited from experiences in dealing with the hazards of radioactive substances of all kinds. The most dramatic achievement during the war — the liberation of atomic energy — has brought with it, and will continue to do so, new hazards to personnel employed in plants where this new force is generated. Safeguards have, on the whole, been unusually effective, and thanks to the war and the preproduction research, industrial hygiene has known methods to protect employees from dangerous exposures.

The social advances proved even greater than those won in the technical field. Experience has taught that mechanical safety devices by themselves are not sufficient to achieve the best results. The human element has also to be considered, and the co-operation of the group and the individual worker enlisted. Thus, a new concept has developed: that of the worker as a "total man" whose mental and emotional traits and responses are as important for high productivity as his physical capabilities. Since these emotional responses depend not only on working environment but also on conditions outside the working place, another social development followed logically. Plant services were integrated with community health programs, and preventive medicine has become an important factor in ensuring not only the well-being and happiness of the worker but also an increase in his productivity. Management and labor alike are recognizing more and more the necessity and the value of industrial medical services. During 1946 a constantly growing co-operation of employer and employee in instituting and maintaining an effective in-plant medical department has led to encouraging results. Also, workmen's compensation laws show a growing trend toward liberalization, and the "subsequent injury" law already adopted by thirty-two states is a progressive step in this direction; this law relieves the insurance carrier and the employer from being excessively liable when employing a handicapped worker, veteran or nonveteran.

In the educational field, industrial medicine has likewise made valuable advances. Many colleges and universities have recognized this branch of medical science and have instituted special courses to train competent industrial physicians and engineers. The need for providing medical services for the smaller plant has been appreciated, and numerous plans have been suggested by industrial physicians and agencies interested in the field of industrial medicine. One index of the general progress made in the field of industrial medicine is the increased employment of industrial nurses. In 1941 only 6000 nurses were doing industrial work, whereas in 1945 their number had risen to 14,000 — an increase of 133 per cent.

ATOMIC ENERGY AND PHYSICAL AGENTS

The fact that atomic power has been harnessed on an industrial scale for the first time in history brings the possibility of the more widespread industrial uses of atomic energy appreciably nearer and makes it of immediate importance to the industrial physician, who will have to face the new and difficult problems posed by nuclear energy. Bale[1] points out that until recently nuclear energy was produced only in large-scale operations at fixed sites where adequate methods of protection reduced to practically nothing the hazards to personnel. Health reports from Oak Ridge, for example, were uniformly good, and Clarke[2] states that the anxiety that working with radioactive materials might cause sterility is completely refuted by the birth rate at Oak Ridge, which was one of the highest of any city in the country. All these protective measures, however, have been expensive and have required the continued vigilance of the staff charged with the safety of the project.

Atomic energy will presumably be used in industry in nonmobile plants as a source of electricity, and as such it will have to compete economically with electricity derived from water power and from coal-burning installations. Despite the economic

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necessity of cutting costs, the industrial physician and engineer charged with the health and safety of workers, as well as with the health and safety of inhabitants living in the neighborhood of nuclear plants, must insist on adequate protection and safety measures, even if the needed precautions may have to be more elaborate than ordinarily realized.

Dowdy[3] offers the reassurance that no insuperable difficulties stand in the way of adequate protection of workers and public alike, for the types of radiation encountered in the liberation of atomic energy are in the main not new either to physicians or to radiologists and physicists. The radium industry has for many years dealt with and overcome these problems. Only neutron radiation is a completely new type of radiant energy not previously encountered in industry. The enviable record of the Manhattan District Project, however, is ample proof that the hazards of production in this field need be no greater than those in many other industries.

Kennedy[4] likewise states that although the high level of neutron and gamma radiations of nuclear matter represents a real hazard to operators, observers and bystanders and although these hazards, being inherent in the fission reaction, are not subject to elimination from the process, they are definitely subject to careful control.

In the extensive wartime industry of radium dial painting, in which workers were inevitably exposed to radium, work has been done to prevent the fatal anemias and osteosarcomas that claimed many victims after World War I. In a discussion of the medical status of such workers Tabershaw[5] presented a comprehensive survey of 52 radium dial painters who worked a minimum of eighteen months in applying luminous paint. Their physical and dental conditions, their blood counts and their nutritional status were recorded, and the conclusion reached that reliance on medical examination is not sufficient to control the hazard. Only regular breath-radon analyses at intervals of six months or, in certain cases, of even three months, combined with an educational campaign regarding work habits, personal hygiene and care of teeth, can prevent deleterious effects.

Beta and gamma radiation is dealt with in a paper by Evans.[6] In textile and paper mills it is often necessary to install radioactive static eliminators as a safety appliance. These eliminators reduce the hazard of fire or explosion of chemical vapors by eliminating the possibility of electrical sparks due to static electricity. These safety appliances, however, being radioactive, present in themselves a new industrial hazard owing to the penetrating radiation that they may emit. Since the radiations that are generated and the maximum permissible doses are known, it is simply an engineering problem to provide adequate safety measures. Shields, sufficient distance and even magnetic deflection are recommended. The commercial development of alpha-ray sources (plutonium, protactinium or polonium) that, unlike radium, emit few or no beta or gamma rays may in the future become a problem.

Injuries resulting from exposure to radioactive substances and roentgen rays are discussed by Dunlap.[7] The main hazards are encountered in plants where x-ray or radiologic equipment is manufactured or radioactive substances used. Safety measures consist in the elimination of all unnecessary exposures by the use of adequate lead shielding, long-handled instruments, speed in carrying out dangerous operations, the rotation of workers and, finally, careful medical supervision.

Out of Hanford also comes a most interesting paper by Cantril[8] on the use of radioactive tracers in industrial medicine. The method of employing a small quantity of a particular radioactive isotope to trace or tag the chemical flow or metabolic patterns in a chemical or physiologic process is not new and was used in various fields prior to the development of atomic energy. This procedure has been employed not only in physiology but also in industry. In the control of exposure of radium dial painters to radium it was possible to use the radioactive properties of the noxious agents in measuring the quantities of contamination on benches, floors, hands and so forth or in the air. The experienced industrial hygienist will with a little imagination recognize the possibilities of applying these technics to occupational hazards quite unrelated to the field of radioactive materials. A tracer could be found that, by virtue of its radiations, half-life and metabolic properties, would measure contamination of working locations and personnel. The method, however, will have to be controlled, not to substitute a radioactive toxin for a nonradioactive one.

The effects of electric injuries, with particular reference to the nervous system, are considered by Hyslop.[9] Accidental deaths from electricity (not due to lightning) represent 0.9 per cent of all accidental deaths, and about 0.4 per cent of all accidents in industry and in the home. Approximately 17 per cent or about 1 electric accident in 6 is fatal. Vital organs and tissues traversed by the current may be functionally impaired or even destroyed, and although other parts of the body may be affected with resulting functional or structural damage, persisting psychoneurotic reactions, as well as a permanent total disability, are rare. Electricity, in contrast to damage done by radium or radioactive substances, does not cause progressive dissemination or local degenerative structural defects.

The Small Plant

Industrial hygiene and industrial medicine are as necessary for the small plant as for the great industrial concerns. These small plants, however, which Bloomfield[10] calls "the underprivileged members of

the economic system," are often not able to provide an adequate medical department for health protection and health maintenance of their workers. It is essential that the achievements of modern industrial hygiene be made available to the small plant, for two thirds of American workers are employed in such plants. National and community health agencies in various states have therefore united with the small-plant employers and employees to bring about an improvement of worker health through control of his environmental hazards and his individual health problems. Millman et al.[11] report on the work of the Industrial Health Unit of New York City. Seven small plants, varying in size from 70 to 700 employees who had either inadequate or no health service, participated in the program. The results were satisfying. A program of this kind has proved to be economically feasible provided that proper plant organization can be installed and that official as well as unofficial community groups take an active interest in the success of the undertaking.

In California an industrial group — F. M. Poole, M. D., and Associates — has been formed to serve the industries in the growing industrial San Fernando Valley.[12] The staff of the group includes six physicians with extensive industrial experience, and the aim is to develop a comprehensive industrial medical plan designed specifically to benefit a variety of industries and plants of various size, down to the smallest, employing as few as ten workers.

How necessary it is to institute such health services for small plants and to enlist the interest of management, labor and community agencies in creating and maintaining it was proved by a survey conducted by the Bureau of Industrial Health of the State of New Jersey. Hazard[13] states that the results show that only 1 plant out of every 5 employing 101 persons or more and only half the plants employing 501 persons or more had adequate doctor service. Among plants employing 101 or more apiece 54 per cent did not offer physical examinations for either placement or health improvement, 65 per cent had no adequate plant food service, and nearly half — 43 per cent — were without a nurse.

Strong[14] insists that best results can be maintained if plant health service is based on the co-operation of management, labor, public-health agencies and special health agencies, which, in a common effort, can provide the protection and the medical care for the workers that the small plant is economically unable to afford. The installation and maintenance of a first-aid kit under the control of one or more employees, especially trained in first aid technics, is suggested for the small plant, store or shop where only 10 workers or less are employed. Although the careful maintenance of such a first-aid kit is unquestionably most important, however, it is unfortunately true that the indifference of both employer and employees probably makes such a kit often not only useless but a danger instead of a help.

In preventive medicine the small plant is also under a disadvantage compared to the large, industrial concerns. Pollak[15] compares conditions regarding tuberculosis in the Caterpillar Tractor Company of Peoria, Illinois, with those in smaller plants in the same area that do not maintain a full-time medical department. In the survey of 13,547 employees of the Caterpillar Tractor Company, the incidence of active pulmonary tuberculosis was 0.16 per cent. On the other hand, in the smaller plants covered by the same survey, of 12,129 employees given x-ray examinations, the case incidence of active pulmonary tuberculosis was 0.29 per cent, or nearly twice as high as that in the group that enjoyed the industrial health protection of a competent and comprehensive medical service.

The problem of the small plant in industrial medicine, being chiefly an economic one, must be solved by the co-operation of all concerned, including management, labor and the official and non-official health agencies.

Job Placement

During the war, virtually all able-bodied men having been taken into the armed forces, industry had to maintain vital production with a labor supply recruited from the ranks of the physically handicapped, the superannuated and women. It was found, however, that these physically inferior workers could and did achieve an excellent production record. The problem of the physically impaired worker has lost nothing of its urgency and significance in the postwar period.

Out of the war a new concept of industrial physical fitness has evolved: a worker is physically fit if he can do a good day's work. Harvey and Luongo[16] point out that the capacity for work is a relatively untouched problem and that medical science does not yet possess a method of evaluating it correctly. Ultimately, such capacity rests on the reserve compensation that the personality of the disabled worker provides. The examining industrial physician must therefore possess a certain diagnostic flair and must be able to judge the reserve compensation behind the visible fact of physical impairment. Although many doctors have evolved their personal formulas regarding the job placement of the handicapped, illuminating their rules with detailed charts, the insight of the examining physician will in the end prove of greater value than any purely schematic approach. In general, it is recommended that any doubt should be resolved in favor of the applicant, since the desire for the work oftener than not creates the capacity to perform the work. All studies on properly placed handicapped workers indicate that they are on a par with their able-

bodied fellow workers. Training and placing costs are balanced by the fact that the handicapped workers stay on the job longer than the average able-bodied worker.

Back strains, representing one of the most important problems in industrial medicine, have been investigated by Parton and Biram.[17] These authors report on a series of a thousand consecutive routine roentgenograms of the lower back taken as part of a preplacement examination; 502 cases were considered normal, and anomalies were revealed in 498. The authors believe that these deformities play a significant role in the decompensation of backs in the age group over forty-five years, and in spite of the lack of knowledge regarding correlation with job placement, preplacement x-ray examination is useful. These studies are in accord with similar studies by other investigators.

Industry has to deal not only with disabled veterans and workers but also with physically unimpaired workers and their various problems. Ghiselly[18] points out that the basic considerations in job placement and personnel procedures are not forms and schedules but rather men and women who have worked together in a complex social environment. He also insists that job placement is not a static affair but a dynamic process that does not end with the assignment of a particular worker to a particular job. New problems of adjustment, change in conditions and change in personality or attitude will constantly arise and make job placement a continuing process.

Management has often found it advisable to employ a psychiatrist either in an in-plant or in a consultative capacity. Clarke and Law[19] state that a few progressive concerns have always availed themselves of the services of a staff psychiatrist, but during the past few years, psychiatry in the field of industry has made great headway. Psychiatric appraisal of the personality of the workers has been found of great value, not only in job-placement but also to combat absenteeism, undue work termination and accidents, which are often due more to faulty human material than to flaws in the technical department.

Clarke and Law, as well as Leggo et al.[20] reporting on their work in industrial psychiatry done at the Oak Ridge community where the atomic bomb was developed, speak of excellent results among thousands of employees. It was found that psychologic and psychiatric pre-employment screening and the application of psychiatric insight in job assignments gave uniformly good results. Therapy for on-the-job patients who presented emotional disturbances proved especially profitable, for it prevented nondisabling maladjustments from developing into major disabling breakdowns. Leggo et al. also state that during their two years' work at Oak Ridge they almost never encountered a patient with a major psychosis. The bulk of the case load consisted of anxiety states, reactive depressions and psychosomatic reactions. It is safe to assume that similar conditions prevail in industry in general, and that with a minimum of treatment conspicuous on-the-job improvement can be achieved. Such improvement, implying as it does adjustment not only to work conditions but also to home environment, makes the employment of the psychiatrist in the industrial field a gain for the entire community.

Dermatitis

Dermatitis continues to be the persistent problem of industrial hygiene, remaining in the forefront in the incidence of occupational diseases.[21] It is occasionally encountered in unexpected places and from unexpected causes. It has been universally accepted that dermatitis arising from irritant dusts, mists and vapors only develops if the irritating agent comes into actual contact with the skin. Baker and White[22] report a case of occupational dermatitis due solely to the inhalation of trichlorethylene. This solvent vapor, used as a degreasing material, was blown by several large fans directly into the open office of a foreman, who developed a severe dermatitis. To establish that only inhalation and not penetration of the gas through clothing produced the dermatitis, the patient was sent to work wearing a gas mask. When the mask was worn no skin irritation occurred; when the mask was discarded the dermatitis reappeared. Installation of a ventilating system that dispersed the offending fumes through the roof cured the condition. This is the first report of a case of generalized dermatitis produced solely by inhalation.

It is generally accepted that scabies is transmitted only by direct contact. Fifteen women in a statistical department, however, contracted the disease through merely handling reports passed to them by a colleague suffering from the affliction, and the outbreak was deemed both occupational and compensable.[23]

A number of authors discuss the causes of occupational dermatitis. Klauder and Hardy[24] report on an additional 539 cases as a supplement to a previous report of 1113 cases of cutaneous diseases.[25] The review of 2297 cases gives a comprehensive picture of the various causes of occupational dermatoses. These causes are divided into seven groups according to the actual causative irritant and not according to occupation, as is the more usual procedure. The authors believe that the listing by causes helps in suggesting preventive and investigative measures and facilitates teaching of the subject. The irritants resulting most frequently in occupational dermatitis were solutions used in electroplating and compounds of chromium; the highest incidence rate occurred in the printing trades, electroplating, tanning, the manufacture of textiles and dyeing. Primary irritants accounted for 24.8 per cent of cases, and sensitizers for 12.5 per cent.

Other causes were trauma, wet work, solvents, oil and chlorinated hydrocarbons. Lane,[26] in a review of compensation paid for disability due to occupational dermatitis describes a steady increase in the incidence of dermatitis but a reduction in the lost time, average indemnity and cost of medical treatment of these cases. Peck[27] found the chief causes of industrial dermatitis to be petroleum products and greases, alkalies, cement and solvents, in the order listed.

Keil et al.[28] proved by patch test that the same ingredients that produce poison ivy are responsible for the dermatitis caused by cashew-nut-shell liquid. Both plants are members of the same plant family, Anacardiacea, and persons sensitive to one plant are sensitive to the other. Cashew-nut-shell liquid is an agent that causes a great deal of difficulty, especially in stevedoring operations.

Schwartz and his associates[29] have developed an entirely new technic to determine the value of various commercial ointments supposed to withstand penetration by alkali, acid or oil. Three standard substances and twenty-seven commercial products were tested, and on the whole it was found that anhydrous wool fat and white petrolatum are quite as efficient protective measures as the best commercial products. This was true for protection against alkalies and acids, as well as for oil. Other authors observed certain commercial products to be effective. Excellent results have been reported from the use of Paraprol,[30] — a product consisting of urea, lauryl sulfoacetate and benzyl alcohol,— which was tested on 300 employees and found to be effective both as a prophylactic and as a therapeutic measure; no unfavorable reactions from its use have been observed.

Preventive measures from another angle are suggested by Klauder and Brill.[31] They attempted a correlation of the boiling ranges of some petroleum solvents with their dermatologic harmfulness. The irritant actions on the skin of petroleum fractions between kerosene and light spindle oil were dealt with, and the conclusion was reached that kerosene and solvents of lower boiling range invariably exert irritant action. The irritant action definitely decreased as the boiling range increased, and it therefore seems advisable to employ a product of as high a boiling range as possible consistent with the purpose for which it is to be used.

Protective clothing and personal hygiene are still of utmost importance to avoid undue exposure with resulting skin damage. Workers, as well as their foremen, must be educated to make conscientious use of the accepted preventive measures. Some of the methods used in the Army may be helpful in this educational endeavor. Personal or group talks, pamphlets, pictorial presentation and so forth can be utilized to explain and visualize the inevitable results of neglect and the equally indisputable rewards of proper care of the skin.

Specific Chemicals

In dealing with the toxicity of the vast number of chemicals used in industry, the industrial physician is, above all, on the outlook for new and improved means to determine at an early stage the deleterious effects. The thymol barbital test, which is reported by McCord and his co-workers,[32] is therefore of particular interest as a means of detecting toxic effects of carbon tetrachloride. Functional impairment has taken place on levels lower than 100 parts per 1,000,000, and it is even possible that in some cases death occurred from apparently trivial exposures. In 1944 Maclagan,[33] of England, found, in the course of an inquiry on the colloidal-gold reaction of serum, that thymol, introduced for antiseptic purposes into the barbital employed as buffer along with certain serums, led to a turbidity apart from any action of the usual constituents of the colloidal-gold system. Maclagan[34] later established that this turbidity was quantitative and of diagnostic significance for hepatic dysfunction. McCord and his co-workers[32] showed that this test is of value in determining the action of carbon tetrachloride on the liver and other tissues. An investigation was made on 712 hospital patients and 82 normal persons. The results indicated that the quantitative association of increasing turbidity with hepatic injury appears well founded. Exposure to low levels of carbon tetrachloride vapors may cause functional changes and even organic injury. In animals, concentrations of 40, 60 and 80 parts of carbon tetrachloride per 1,000,000 produced thymol turbidity.

Although this work helps to determine minimal injury, the difficulty of measuring carbon tetrachloride vapors in the air has also been practically solved. Schayer and Ackerman[35] report a modified combustion method as used by the Army Industrial Hygiene Laboratory that permits the rapid and efficient determination of chlorinated hydrocarbon in air. Glass gas-sampling bulbs of capacities of 275 to 500 cc. were used, and the collected vapors were burned in a modified sulfur lamp. The amount of hydrogen chloride formed was turbidimetrically determined with the help of a spectrophotometer. Four chlorinated hydrocarbons were studied, among them carbon tetrachloride, and it was found that concentration of vapor can be determined with an accuracy of 88 to 90 per cent.

Nutritional factors in industrial poisoning have been of interest for some time, especially regarding liver disease. Shaffer, Carpenter and Moses[36] report that, although methionine is of undoubted therapeutic and prophylactic value in the protection of protein-deficient animals against carbon tetrachloride poisoning, it exerts no protective action against the liver damage of normal animals poisoned with carbon tetrachloride.

Volatile hydrides that have toxicologic importance are dealt with by Webster.[37] Whereas many of these compounds have no industrial application, it is quite likely that the rapidly increasing production of the rarer inorganic chemicals will lead to the use of a number of volatile hydrides. Many of these compounds are of an insidious character, entering the body through inhalation, and it is necessary to recognize the potential danger from breathing the substances.

An extensive study of the effects of beryllium compounds is offered by Hardy and Tabershaw.[38] Seventeen cases of a puzzling disease, which the authors call "delayed chemical pneumonitis," appeared among the employees of a firm manufacturing fluorescent lamps. In the course of the manufacture beryllium compounds were used, and although no clear etiology has been established, the weight of evidence indicates that beryllium was responsible. The fact that the 17 workers affected were employed in one building during the same period clearly points to common exposure. The clinical features of the disease are a delayed onset, intense dyspnea, marked loss of weight and unfavorable prognosis. The relation of the acute form of this disease to the more chronic type has not yet been defined.

The increased use of tellurium in industry has awakened a deeper interest in the problem of its effect on the organism. De Meio[39] reports a series of animal experiments in which tellurium was fed to rats. Although the low toxicity of elementary tellurium, as opposed to tellurate and tellurite, was again established, it seems that long exposure to tellurium in great concentration tends to lower normal increase in weight and growth.

Ethylene dichloride is a chlorinated carbon that is widely used as a solvent and for other industrial purposes. An extensive literature dealing with its toxicity, its peculiar ability to render the cornea turbid and the effects of various protective agents already exists. Heppel et al.[40] report the effects of daily inhalations of this gas on a variety of animals. The results indicate that dichloroethane is one of the more toxic of the frequently used hydrocarbons and should be employed only when proper precautions are taken.

Ketones, alcohols, esters, ethers and miscellaneous solvents, all used widely in industry, are the subject of a study by Silverman and his associates[41] in which sensory responses to these vapors were evaluated. Sensory limits for eighteen organic solvents were established, and since eleven of these solvents had no previous evaluation of permissible concentration, the data offered are of special interest to the industrial hygienist and engineer.

The report of a case of arsine poisoning by Hawlick and Ley[42] should be mentioned. Freshly diluted hydrochloric acid, which had been treated with an inhibitor containing a soluble arsenic compound, was poured through a water jacket. The operation was carried out in a poorly ventilated room, and precautions against inhalations of fumes were not observed. The employee exposed to the fumes experienced after a few hours severe hemoglobinuria, prostration, jaundice, an enlarged and tender liver, peripheral neuritis and distinct hemolytic anemia. The tentative diagnosis of acute hemolytic anemia secondary to exposure to a heavy metal, possibly a compound of arsenic (arsine), was confirmed when a specimen of the metal pipe through which the acid had been poured was tested by the Army Industrial Hygiene Laboratory.

An effective agent against industrial poisoning with arsenic and mercury has been discovered in BAL (British antilewisite). This compound, which was developed during the war as an antidote for lewisite, has proved successful in the treatment of arsenic poisoning of any type. The drug (2-3 dimercaptopropanol) is the best from the standpoint of skin penetration when used as an ointment. Although the least dangerous of the thiols, it is quite toxic, and its use must be carefully controlled.

Miscellaneous Conditions

Penicillin in two different forms has been found highly effective in group medicine in both the Army and the Navy. Agmar[43] reports a study in which crude penicillin was inoculated into surgical-gauze dressings. This preparation proved dramatically effective for such minor infections as boils, carbuncles, secondary epidermophytosis of the feet, eye and ear infections, chronic ulcers of the leg and infected cyst. Crude penicillin solution and penicillin gauze did not cause any reactions and worked more rapidly than the sulfonamides. These preparations are easy to make, readily transportable, simple to use and markedly potent.

Levitt and Leathen[44] are equally enthusiastic about penicillin lozenges in the treatment of oral infections. One hundred and seventeen patients with acute infection of the nasopharynx and tonsils were treated with penicillin lozenges of 500-unit strength each. Results of the treatment were excellent. Of the 117 patients, 110 were cured rapidly, 87 of them on an outpatient status. Only 7 patients did not respond to the treatment. Five of these cases had tonsillitis complicated by cervical adenitis, which evidently proved that local concentrations of penicillin cannot penetrate tonsillar crypts or reach infections deep in the tissue of lymph nodes.

A program of malarial surveillance for the industrial physician is offered by Shillito.[45] Industrial concerns expanding their interests and activities into tropical countries and war veterans returning from these regions make it necessary for a medical department to become familiar with effective methods of handling this problem. Two possible courses are recommended: either a careful surveillance without drug therapy or a course of daily doses of quinacrine

hydrochloride (0.1 gm. in one tablet) for thirty days. Quinine sulfate may be substituted if gastrointestinal disturbances occur. A definite diagnosis should never be made until confirmed by laboratory findings; treatment should then be immediately instituted.[46]

Comprehensive articles describing the more frequent manifestations of caisson disease, such as pain occurring near joints (bends), respiratory discomfort (chokes), cutaneous lesions and abdominal gaseous distentions, have been published. Lund et al.[47] describe a case of severe decompression illness of several days' duration. The patient, a twenty-year-old student, had with thousands of others volunteered for decompression tests. Ascent was made to 38,000 feet. Several hours after decompression, severe symptoms referable to the central nervous system occurred. A gradual return to normal took place over a period of several days, but slight neurologic residua persisted for approximately two and a half months. No explanation is offered for the mechanism in this condition, but it is clear that there must be some agent or mechanism following decompression that is capable of local action on the central nervous system.

Surgical removal of lumbar herniations of intervertebral disks should be theoretically of great relief to sufferers of low backache and sciatica. Indeed, White and Peterson[48] present statistics on a series of 39 naval patients operated on with removal of a definite disk herniation, and these data show excellent results. In the 39 patients, late end results (from five months up to two years) are known for 23 men. Of these, 13 have returned to active duty or their former jobs, 4 are back on limited duty with a fair prospect of an ultimate return to full activity, 1 is engaged in moderately active work, and 5 are undergoing or likely to require further investigation because of a return of symptoms. Thus, 75 per cent of patients have been restored to full or limited duty, and this compares favorably with the results previously achieved. These good results are credited mainly to the preoperative technic. Only patients who, while suffering from severe low backache and sciatica, had failed to respond to conservative orthopedic treatment of at least a month's duration were held eligible. In addition to the classic signs of compression, almost all patients showed a definite filling defect with Pantopaque. Whether these results can be duplicated in civilian practice remains to be seen, but since backache is one of the problems of industrial medicine, the success of White and Peterson is of general interest.

* * *

In dealing with occupational medicine in retrospect and prospect Newquist[49] asserts that although much has been accomplished much remains yet to be done. To raise the general physical fitness of the industrial population, to rehabilitate the disabled war veteran and to enhance the safety of the industrial environment will be the endeavor of every industrial physician. Every new day, however, brings new challenges. The entrance of atomic power into the industrial field, which is now an accomplished fact, brings with it hazards whose magnitude cannot be quite foreseen. What can be foreseen, however, is that the hazards will be overcome and the dangers circumvented.

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-SIXTH ANNIVERSARY

May 20, 21, and 22, 1947

THE one hundred and sixty-sixth anniversary of the Massachusetts Medical Society was observed at the Hotel Statler in Boston on May 20, 21 and 22, 1947.

Fifteen hundred and twenty-one physicians, one hundred ladies and two hundred and ninety-seven exhibitors were registered.

The length of the meeting was increased this year by beginning the sessions in the morning of the first day instead of in the afternoon. The Supervising Censors met in Parlor C at 4:30 p.m. on the afternoon of the previous day, May 19. This meeting was followed by the Cotting Supper in Parlors A and B, which was attended by 205 councilors. The annual meeting of the Council was held in the Georgian Room at 7:00 p.m. with 189 councilors in attendance, as recorded in the attendance books.

Tuesday, May 20

The first general session, a symposium on industrial medicine, opened at 9:00 a.m. in the Georgian Room under the co-chairmanship of Dr. Daniel L. Lynch and Dr. Gerald L. Doherty.

The one hundred and sixty-sixth annual meeting of the Society was held in the Georgian Room at 11:00 a.m., President Dwight O'Hara presiding. The attendance was about 450. Dr. O'Hara spoke on "The State of the Society," after which the annual oration, "Medical Care in Our Free Society," was delivered by Dr. Leland S. McKittrick. The annual luncheon was served in Parlors A, B and C to 101 fellows.

The second general session, a symposium on medical care for the people of Massachusetts, was held in the Georgian Room at 2:00 p.m. under the co-chairmanship of Dr. Reginald Fitz and Dr. Albert A. Hornor. The Shattuck Lecture was delivered at 8:00 p.m. by Dr. William Dock, of Brooklyn, New York, on the subject "Clinical Significance of Circulatory Peculiarities of Some of the Vital Organs." (This lecture appeared in the May 22, 1947, issue of the *Journal*.)

Wednesday, May 21

The third general session was held at 9:00 a.m. under the co-chairmanship of Dr. Isaac S. F. Dodd and Dr. Allen G. Rice.

At noon certain of the sections held their luncheons, followed by their annual meetings. The Section of Medicine, under the chairmanship of Dr. Francis C. Hall, met in Parlor A. The attendance was 110. The Section of Pediatrics, under the chairmanship of Dr. Hyman Green, met in Parlor B, with 60 members in attendance at the luncheon and 84 at the scientific meeting following. The Section of Dermatology and Syphilology met in Parlor C under the chairmanship of Dr. Jacob H. Swartz, with 43 in attendance at the luncheon and 55 at the

meeting. The Section of Physiotherapy met in the Hancock Room under the chairmanship of Dr. William D. McFee, with 15 members present.

At 2:00 p.m. the fourth general session, a symposium on bronchiectasis, was held in the Georgian Room under the co-chairmanship of Dr. Richard H. Sweet and Dr. Louis H. Nason.

The annual dinner was held in the Georgian Room at 7:00 p.m., with 418 in attendance. The speaker of the evening was Dr. Harrison H. Shoulders, president of the American Medical Association, who delivered an address entitled "The Responsibilities of the Medical Profession."

Thursday, May 22

The fifth general session was held in the Georgian Room at 9:00 a.m. under the joint chairmanship of Dr. Franklin G. Balch, Jr., and Dr. Walter E. Garrey.

At noon the remaining scientific sections held their luncheons and meetings. The Section of Surgery under the chairmanship of Dr. Alexander J. A. Campbell met in the Salle Moderne, with 100 in attendance. The Section of Obstetrics and Gynecology under the chairmanship of Dr. William J. McDonald met in Parlor A, with 60 in attendance. The Section of Radiology under the chairmanship of Dr. Edward B. D. Neuhauser met in Parlor C, with 39 in attendance. The Section of Anesthesiology under the chairmanship of Dr. Sidney C. Wiggin met in Parlor B, with 42 members attending.

The sixth general session was held at 2:00 p.m. under the joint chairmanship of Dr. Howard F. Root and Dr. Gordon M. Morrison.

There were eighty-nine technical and four scientific exhibits.

On Wednesday, May 21, at 2:45 p.m. the Massachusetts Medico-Legal Society held its meeting concurrently in Parlor C.

At the annual golf tournament held at the Woodland Golf Club on May 20, the low gross winner was Dr. R. J. Nugent, the second gross winner, Dr. M. Sargent, the winner of the low net, Dr. A. D. Crowell, and the winner of the second low net, Dr. G. Quigley. An exhibition of works of art by members of the Massachusetts Physicians' Art Association was on view throughout the meeting.

The special list of officers, standing and special committees, councilors, censors, admissions and deaths is appended.

Joseph Garland, *Secretary*

Annual Meeting of the Society

The one hundred and sixty-sixth annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. Dwight O'Hara, in the Georgian Room of the Hotel Statler, Boston, at 11:00 a.m., May 20, 1947. Approximately 450 fellows were present.

The Secretary submitted the record of the last annual meeting held on May 22, 1946, as published in the September 19, 1946, issue of the *New England Journal of Medicine*. It was moved and seconded that the record be accepted, and it was so ordered by vote of the fellows present.

The Secretary reported that the membership of the Society as of May 22, 1946, was 5989, and as of May 20, 1947, it was 6256. During the year there had been a loss of 140 fellows — 95 by death, 42 by resignation and 3 by deprivation of membership. There had been a gain of 407, of which 403 were new members and 4 had been reinstated. It was moved and seconded that the report be accepted, and it was so ordered by vote of the fellows.

At the request of the President, the vice-president, Dr. Isaac S. F. Dodd, assumed the chair, and Dr. O'Hara gave the following address.

One may come to the office of president-elect and later president of the Massachusetts Medical Society supposing himself to be somewhat familiar with its routine affairs. But when one finds himself an *ex-officio* member of thirty-odd committees and begins to see the various ways in which their many interests cross, he can but stand and marvel at the volume and the continuity of the work accomplished year after year in the name of the Society. It is an impressive, enlightening and pleasant experience. After a preliminary initiation the mechanism of the Society begins to reveal itself; there are a faithful few who toil at necessary basic tasks — our late lamented secretary, Dr. Michael A. Tighe; our treasurer, Dr. Eliot Hubbard; our executive secretary, Mr. Robert St. B. Boyd; our editor, Dr. Robert N. Nye with his able assistants and their stenographic staff; these turn the wheels, gears and cogs of the Massachusetts Medical Society.

During the past year the machinery has needed some internal adjustments. In October we lost by illness the services of Miss Katharine Cowles, who had long kept our clerical house in order; in January we took over, by previous arrangement, our own office obligations and thus ended a period of years in which we had been dependent on the staff of the *New England Journal of Medicine* for whatever bookkeeping and stenography were needed; in January we were first deprived of the services of our faithful secretary, though, happily we thought, he was able to direct us from afar while his coronary vessels rebuilt a collateral circulation. But this was not to be. He was able to make a few more visits to the headquarters of the Society, and attended several committee meetings during the early spring. On April 8, the day before we expected him to join us again at 8 Fenway, Dr. Tighe suddenly died.

We were warned but not prepared for this. Along with his sense of humor, his kindly comradeship and his interest in every ramification of the Society, Dr. Tighe has taken with him a knowledge of our affairs which surpassed that of any other man. These are the great losses of every organization — the contemporary intellects on which we depend, even thoughtlessly as we depend on roadways and bridges to get us about our daily work. We come to take them for granted, and then they are gone. This was an irreparable loss. I ask the Society to stand for a moment of silent tribute to Secretary Michael A. Tighe.

In the face of these adjustments the Society has been fortunate in its office personnel, including among others Miss Margaret E. Biggy, Miss Elinor Kelley and, especially, Mr. Boyd. How confidently a troubled institution may depend on the character and fidelity of a few individuals was vividly demonstrated to those of us who were close to these changes, and were naturally anxious to have the engines working smoothly while we were taking our turn at the steering wheel.

The one hundred and sixty-sixth year of the Massachusetts Medical Society is now closing. An organization which has survived over such a period of time not only

has inevitable traditions and duties to which it must respond but also finds itself inevitably watched by other groups in the larger community of which it is a part. It must solve its problems not in its own interests alone but in the interests of the people as a whole. This is peculiarly true in our profession, and it is on this account that we tend to come to our decisions slowly. Much spade work must precede the actual conclusion of most of our business, and much spade work is now quietly going on, the result of which may not become apparent for months or years to come. In giving an account of business concluded by the Society, I therefore ask you to bear in mind that none of it is the fruit of a single year of effort, but is rather the natural conclusion of processes which had been initiated and prepared in preceding years. In like manner new business has been proposed and studied during the current year which can only be reported as in progress at the present moment.

Veterans' Contract

Late in 1945 the Veterans Administration evolved a new medical policy, under Generals Bradley and Hawley. The Administration's new policy had two main objectives — the staffing of its expanding hospital facilities, which was placed upon the shoulders of the Nation's medical schools, and the provision of home and office care for all veterans by doctors of their own choosing. Early in 1946 the Veterans Administration asked the Massachusetts Medical Society to undertake the responsibility for the latter function, and a committee under the chairmanship of Dr. Humphrey L. McCarthy undertook what was to become a long and difficult task of orientation and negotiation. The Council devoted a special eight-hour meeting to the subject on April 10 and discussed it again on October 2, 1946. On October 26, agreements based on these discussions were reached with the Veterans Administration, and the matter was placed in the hands of the Blue Shield for administrative arrangements. The contract was signed and went into effect as of December 27, 1947, only to encounter the obstacle that the Blue Shield needed permissive legislation in order to carry out the contract! Opposition to such legislation has since appeared, and there have arisen other complications the details of which are not pertinent at this time. It should be emphasized here that the Massachusetts Medical Society is in no way compromised by these complications because it has sought and seeks nothing in the whole matter. The Society has merely responded and will continue to respond to every suggestion that it might be helpful to the interests of the veterans of this Commonwealth.

Director of Medical Information and Education

During 1945 several of the committees of the Society realized that it needed a more dynamic approach in its relations with the people, their legislators and their spokesmen; we were being socially misrepresented and misunderstood at a time when there was every reason for us to assert ourselves. A bill to license chiropractors reached Governor Tobin's desk; a nation-wide antivivisection effort vented itself in this state, among others; our motives in working for the establishment of the Medical Approving Authority were questioned; the call for postgraduate medical education was mounting beyond all available resources in the Commonwealth. These things led three of our committees to indicate their belief that the time had passed when the Society could depend on its committee members to meet without further aid the challenges of the day. President Fitz skillfully focused these needs for action with the result that the Council, on October 3, 1945, created a position to be known as Director of Medical Information and Education, and agreed to appoint such an officer on proposal by a special group, the Committee of Seven, named for the purpose by Dr. Fitz. Finding an incumbent worthy of so great a task proved to be a difficult matter. The Committee of Seven worked for more than a year before they finally proposed to the Council, on February 5, that Dr. John F. Conlin be named. Dr. Conlin is to begin his duties on July 1, and we bespeak for him the interest and support of every fellow of the Society.

Annual Assessment

The employment of the Director of Medical Information and Education will throw the budget of the Society out of balance. The narrow margin by which we have been able to do business in recent years has been frequently commented on, and was brought before the Council with a proposal for increasing the annual assessment three years ago. At that time and since, certain unmet obligations of the Society have been discussed. A year ago the Council ordered the appointment of a special committee to study the entire problem and to report with recommendations. This report was placed before the Council on February 5, with recommendations of far-reaching importance to the future of the Society and the medical profession in the Commonwealth.

The committee recommended in principle that the Society employ a full-time secretary, that the Society support and thereby expand the usefulness of the Boston Medical Library, that financial support be arranged for needy widows and orphans of physicians, as well as for physicians themselves, that a retirement plan be established for full-time workers of the Society and that our present inadequate headquarters be expanded to provide at least sufficient committee rooms for the proper conduct of our business. The committee recommended an increase in the annual assessment from ten dollars to between twenty and twenty-five dollars. After approving the tentative program as outlined and carefully considering its implications in future expenditure, the Council voted on February 5 that the assessment be increased from ten to twenty-five dollars, beginning on January 1, 1948. In taking this action the Council extended our horizon beyond any to which we have been accustomed. The Society can now gaze out upon a field in which literary and sociologic responsibilities are accepted along with the scientific, educational and political interests of the past. If the world is to have a future it is apparent that the Massachusetts Medical Society intends to make itself a part of it.

Postgraduate Education

Over a period of years the Society has expressed an increasing interest in postgraduate medical education. This has been largely due to the excellent work of a committee under the chairmanship of Dr. W. Richard Ohler. It has sometimes worked as a special committee and sometimes as a subcommittee under another name, such as medical defense, war participation or postwar planning. Whatever it has been called, it has done its job so well that each year our educational activities have been expanded, the high point having been reached in March and April of this year when more than 1200 doctors registered for forty-eight hours of instruction at Sanders Theater in Cambridge. Because this course alone was the equivalent of a complete college education for a score or more men we have been referring to Dr. Ohler as "President of Sanders College" and to Dr. Lewis M. Hurxthal as "Dean." Their curriculum included everything except football, and if it continues to increase in popularity they will be forced to adopt some athletic tactics in order to handle the crowds. In addition to these two there are of course scores of others who have willingly served, not only on the faculty of "Sanders College" but at the district meetings throughout the Commonwealth, at the meetings of the Postgraduate Assembly and on the programs now taking place here at the annual meeting. Altogether a formidable amount of postgraduate medical education is being provided for the doctors of Massachusetts by the fellows of the Society.

The Committee on Postgraduate Medical Education also has continued to operate the Bureau of Medical Information, which has been ably handled by Miss Mary D. Gaston at the Society Headquarters. This activity admirably met an early postwar need. What its future usefulness may be, however, is uncertain; we no longer have great numbers of medical strangers passing briefly through our midst; doctors are becoming established and tend to acquaint themselves individually with the local clinics and opportunities. The monthly cataloguing of the greater Boston clinics is a helpful service, but to continue to operate the Bureau for this activity alone seems a little expensive.

The State of the Society

When I assumed the office of president a year ago, I received felicitations from a friend who pointed out that he had just completed fifty years of uninterrupted fellowship in the Massachusetts Medical Society. It occurred to me that such an event is significant in the life of any man or woman, and is worthy of more than passing notice. Becoming interested, I drew up a list of the fellows who were admitted to the Society in 1897; twenty-two of these were still living. They had obviously passed the age at which they were entitled to retire by our by-laws, yet by consulting the records I found that eleven of them had retained continuous active fellowship over the period of fifty years. I have written to each of these eleven, hoping they could attend their fiftieth meeting; I now propose to read their names, and if they are present I ask them to acknowledge that fact by rising.

Dr. George A. Boucher, of Brockton
Dr. John E. Fish, of Canton
Dr. Arthur P. George, of Haverhill
Dr. Joseph B. Howland, of Hingham
Dr. Joseph I. Lindsay, of Grafton
Dr. James F. Loughran, of Lowell
Dr. Franklin S. Newell, of Boston
Dr. Ernest D. Pillsbury, of Stoughton
Dr. Thomas B. Shaw, of Worcester
Dr. John B. Thomes, of Pittsfield
Dr. William J. Walton, of Dorchester

We have no official award that we can make to these gentlemen, but I extend to them the hearty congratulations of the Society and I present their names to you as tangible evidence that the state of the Society is vigorous and sound.

Dr. George A. Boucher, of Brockton, was present, and acknowledged the greeting of the Society.

The President resumed the chair, and as the next order of business introduced Dr. Harrison H. Shoulders, president of the American Medical Association.

Dr. O'Hara then introduced the delegates who were present from the other New England state medical societies — Dr. Carl T. Phillips from Connecticut, Dr. George E. Young from Maine and Dr. Emery T. Porter from Rhode Island. Each acknowledged this introduction, and Dr. Porter spoke as follows:

Dr. O'Hara, if your members have been reading the papers lately, they have probably seen that Governor Bradford has recently called attention to the fact that there is an old Massachusetts law which states that anybody coming from Rhode Island and Providence Plantations, the home of Roger Williams, is subject to death on crossing the border into Massachusetts.

So I wormed my way through the park this morning, and did not get caught, and I welcome this chance to bring greetings from the State of Rhode Island, where the Rhode Island Medical Society has just finished its one hundred and thirty-sixth annual meeting.

The President next introduced the incoming officers, after which the one hundred and thirty-ninth annual oration, "Medical Care in Our Free Society," was delivered by Dr. Leland S. McKittrick. (The oration appeared in the June 19, 1947, issue of the *Journal*.)

Dr. O'Hara declared the one hundred and sixty-sixth annual meeting of the Massachusetts Medical Society adjourned at 1:00 p.m.

JOSEPH GARLAND, *Secretary*

APPENDIX

OFFICERS FOR 1947–1948

President: Edward P. Bagg, Holyoke, 207 Elm St.
President-Elect: Daniel B. Reardon, Quincy, 1186 Hancock St.
Vice-President: Charles J. Kickham, Brookline. Office, Boston, 508 Commonwealth Ave.
Secretary: Joseph Garland, Brookline. Office, Boston (15), 8 Fenway.
Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland St.
Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave.
Orator: Allen S. Johnson, Springfield, 276 Bridge St.

COMMITTEES ELECTED BY THE DISTRICTS

Executive Committee of the Council — Established 1941 (members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).

PRESIDENT: Edward P. Bagg, Holyoke, 207 Elm St.
PRESIDENT-ELECT: Daniel B. Reardon, Quincy, 1186 Hancock St.
VICE-PRESIDENT: Charles J. Kickham, Brookline. Office, Boston, 508 Commonwealth Ave.
SECRETARY: Joseph Garland, Brookline. Office, Boston (15), 8 Fenway.
TREASURER: Eliot Hubbard, Jr., Cambridge, 29 Highland St.

Term Expires 1948

ESSEX SOUTH: Walter G. Phippen, Salem, 31 Chestnut St. (Alternate: Bernard Appel, Lynn, 281 Ocean St.)
HAMPSHIRE: Henry A. Tadgell, Belchertown, Belchertown State School. (Alternate: Lawrence N. Durgin, Amherst, 66 Amity St.)
MIDDLESEX SOUTH: Harold G. Giddings, Newton Centre. Office, Boston (16), 270 Commonwealth Ave. (Alternate: Arthur M. Jackson, Everett (49), 512 Broadway.)
NORFOLK SOUTH: Fred A. Bartlett, Wollaston (70), 308 Beale St. (Alternate: Harry Braverman, Quincy (69), 43 School St.)
SUFFOLK: Alexander J. A. Campbell, Boston (15), 520 Commonwealth Ave. (Alternate: Howard F. Root, Boston (15), 81 Bay State Rd.)
WORCESTER: Bancroft C. Wheeler, Worcester, 27 Elm St. (Alternate: Franklyn P. Bousquet, Worcester, 390 Main St.)

Term Expires 1949

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Paul P. Henson, Hyannis, 149 Main St.)
BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar St. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect St.)
BRISTOL SOUTH: Richard B. Butler, Fall River, 278 North Main St. (Alternate: Curtis C. Tripp, New Bedford, 416 County St.)
ESSEX NORTH: Rolf C. Norris, Methuen, 247 Broadway. (Alternate: George J. Connor, Haverhill, 81 Merrimack St.)
MIDDLESEX EAST: Kenneth L. Maclachlan, Melrose, 1 Bellevue Ave. (Alternate: Justin L. Anderson, Reading, 53 Woburn St.)
PLYMOUTH: George A. Moore, Brockton, 167 Newbury St. (Alternate: John C. Angley, Bryantville, School St.)

Term Expires 1950

BERKSHIRE: John Hughes, Pittsfield, 74 North St. (Alternate: Edward Wyman, Great Barrington, 244 Main St.)
FRANKLIN: Lawrence R. Dame, Greenfield, 78 Federal St. (Alternate: Frank W. Dean, East Northfield, 185 Main St.)
HAMPDEN: Archibald J. Douglas, Westfield, 30 Court St. (Alternate: Frederic Hagler, Springfield, 20 Maple St.)
MIDDLESEX NORTH: William M. Collins, Lowell, 174 Central St. (Alternate: Artemas J. Stewart, Lowell, 310 Merrimack St.)

NORFOLK: Charles J. E. Kickham, Jamaica Plain. Office, Brookline, 1101 Beacon St. (Alternate: Carl Bearse, Boston, 483 Beacon St.)
WORCESTER NORTH: John J. Curley, Leominster, 89 West St. (Alternate: George P. Keaveny, Fitchburg, 62 Fox St.)

Committee on Nominations — Established 1874 (one councilor and alternate elected yearly by each district medical society).

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Paul P. Henson, Hyannis, 149 Main St.)
BERKSHIRE: John Hughes, Pittsfield, 74 North St. (Alternate: N. Newall Copeland, Pittsfield, 131 North St.)
BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar St. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect St.)
BRISTOL SOUTH: Henry Wardle, Fall River, 173 Purchase St. (Alternate: Donald R. Mills, Edgartown, Box 654.)
ESSEX NORTH: Charles F. Warren, Amesbury, 155 Main St. (Alternate: Percy J. Look, Andover, 115 Main St.)
ESSEX SOUTH: Peer P. Johnson, Beverly, 1 Monument Square. (Alternate: O. S. Pettingill, Middleton, Essex Sanatorium.)
FRANKLIN: John E. Moran, Greenfield, 15 Franklin St. (Alternate: Warren D. Thomas, Montague, Central St.)
HAMPDEN: George L. Schadt, Springfield, 44 Chestnut St. (Alternate: George L. Steele, Springfield, 20 Maple St.)
HAMPSHIRE: Henry A. Tadgell, Belchertown, Belchertown State School. (Alternate: Lawrence N. Durgin, Amherst, 66 Amity St.)
MIDDLESEX EAST: Edward M. Halligan, Reading, 37 Salem St. (Alternate: John M. Wilcox, Woburn, 6 Bennett St.)
MIDDLESEX NORTH: William F. Ryan, Lowell, 219 Central St. (Alternate: William M. Collins, Lowell, 174 Central St.)
MIDDLESEX SOUTH: Harold G. Giddings, Newton Centre. Office, Boston, 270 Commonwealth Avenue. (Alternate: Fred R. Jouett, Cambridge, 1 Craigie St.)
NORFOLK: Albert Ehrenfried, Brookline. Office, Boston (15), 520 Commonwealth Ave. (Alternate: Carlton E. Allard, Dorchester, 428 Columbia Rd.)
NORFOLK SOUTH: Henry A. Robinson, Hingham, 205 North St. (Alternate: Frederick Hinchliffe, Cohasset, 117 South Main St.)
PLYMOUTH: Bradford H. Peirce, South Hanson, Plymouth County Hospital. (Alternate: Alfred L. Duncombe, Brockton, 38 Winthrop St.)
SUFFOLK: Albert A. Hornor, Boston (15), 319 Longwood Ave. (Alternate: Conrad Wesselhoeft, Boston, 315 Marlboro St.)
WORCESTER: Ralph S. Perkins, Worcester, 10 Hackfeld Rd. (Alternate: Franklyn P. Bousquet, Worcester, 390 Main St.)
WORCESTER NORTH: John J. Curley, Leominster, 89 West St. (Alternate: James V. McHugh, Leominster, 55 West St.)

Committee on Public Relations — Established 1939 (one councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively; and the vice-president and secretary of the Society are members ex-officiis).

BARNSTABLE: Paul P. Henson, Hyannis, 149 Main St.
BERKSHIRE: Patrick J. Sullivan, Dalton, 417 Main St.
BRISTOL NORTH: Milton E. Johnson, Attleboro, 33 Bank St.
BRISTOL SOUTH: Harold E. Perry, New Bedford, 159 Cottage St.
ESSEX NORTH: Harold R. Kurth, Lawrence, 57 Jackson St.
ESSEX SOUTH: Bernard Appel, Lynn, 281 Ocean St.
FRANKLIN: Howard M. Kemp, Greenfield, 42 Franklin St.
HAMPDEN: Frederic Hagler, Springfield, 20 Maple St.
HAMPSHIRE: Joseph R. Hobbs, Williamsburg, Main St.
MIDDLESEX EAST: Milton J. Quinn, Winchester, 44 Church St.
MIDDLESEX NORTH: Samuel A. Dibbins, Lowell, 528 Andover St.
MIDDLESEX SOUTH: Ralph H. Wells, Lexington, 1430 Massachusetts Ave.
NORFOLK: Dean S. Luce, Canton, 553 Washington St.
NORFOLK SOUTH: Henry A. Robinson, Hingham, 205 North St.
PLYMOUTH: Charles D. McCann, Brockton, 12 Cottage St.
SUFFOLK: Howard F. Root, Boston, 81 Bay State Rd.
WORCESTER: Nicholas S. Scarcello, Worcester, 1 Sheldon St.
WORCESTER NORTH: James V. McHugh, Leominster, 55 West St.

Subcommittees of Committee on Public Relations

TAX-SUPPORTED MEDICAL CARE — Established 1940.
Thomas Hunter (Worcester), *chairman*; Frederick S. Hopkins, Hampden; Albert A. Hornor, Suffolk; William J. Pelletier, Franklin; and Frank W. Snow, Essex North.

COMMITTEE TO MEET WITH THE MEDICAL ADVISORY COMMITTEE OF THE INDUSTRIAL ACCIDENT BOARD — Established 1942.
Gordon M. Morrison (Middlesex South), *chairman*; Charles H. Bradford, Suffolk; Joseph H. Burnett, Middlesex South; Somers Fraser, Suffolk; and William W. Teahan, Hampden.

POSTPAYMENT MEDICAL CARE — Established 1942.
Norman A. Welch (Norfolk), *chairman*; Fred A. Bartlett, Norfolk South; Michael F. Barrett, Plymouth; James H. Brewster, Bristol North; James T. Brosnan, Worcester; Lucien R. Chaput, Essex North; Charles F. Fasce, Berkshire; Patrick E. Gear, Hampden; Loring Grimes, Essex South; Francis T. Jantzen, Suffolk; Egon E. Kattwinkel, Middlesex South; Howard M. Kemp, Franklin; William B. LeBrecht, Worcester North; Wilfred L. McKenzie, Middlesex East; Harold E. Perry, Bristol South; Henry A. Robinson, Norfolk South; Harold F. Rowley, Barnstable; and Elmer E. Thomas, Hampshire.

Committee on Legislation — Established 1942 (one councilor elected yearly by each district medical society).

BARNSTABLE: Julius G. Kelley, Pocasset, Barnstable County Sanatorium.
BERKSHIRE: John Hughes, Pittsfield, 74 North St.
BRISTOL NORTH: William M. Stobbs, Attleboro, 63 Bank St.
BRISTOL SOUTH: Curtis C. Tripp, New Bedford, 416 County St.
ESSEX NORTH: Nicandro F. DeCesare, Methuen. Office, Lawrence, 57 Jackson St.
ESSEX SOUTH: Loring Grimes, Swampscott, 84 Humphrey St.
FRANKLIN: Harold R. Mahar, Orange, 1 High St.
HAMPDEN: Arthur H. Riordan, Indian Orchard, 147 Oak St.
HAMPSHIRE: Lawrence N. Durgin, Amherst, 66 Amity St.
MIDDLESEX EAST: John M. Wilcox, Woburn, 6 Bennett St.
MIDDLESEX NORTH: A. Warren Stearns, Billerica.
MIDDLESEX SOUTH: Kenneth J. Tillotson, Belmont. Office, Waverley, McLean Hospital.
NORFOLK: Solomon L. Skvirsky, Chestnut Hill. Office, 546A State House, Boston.
NORFOLK SOUTH: David L. Belding, Hingham, 215 Main St.
PLYMOUTH: Alfred L. Duncombe, Brockton, 38 Winthrop St.
SUFFOLK: William E. Browne, Boston (15), 587 Beacon St.
WORCESTER: George R. Dunlop, Worcester, 53 Massachusetts Ave. (chairman).
WORCESTER NORTH: John J. Curley, Leominster, 89 West St.

Subcommittee of the Committee on Legislation

NATIONAL LEGISLATION — Established 1946.
Elmer S. Bagnall (Essex North), *chairman*; David L. Belding, Norfolk South; Vlado A. Getting, Middlesex South; Donald Munro, Suffolk; and Augustus Thorndike, Suffolk.

STANDING COMMITTEES FOR 1946–1947

ELECTED BY THE COUNCIL, MAY 19, 1947

	Date of Appointment
Publications — Established 1825.	
Richard M. Smith, Suffolk	June 6, 1933 (appointed chairman May 21, 1941)
Oliver Cope, Middlesex South	May 21, 1941
John Fallon, Worcester	November 14, 1944
James P. O'Hare, Suffolk	June 9, 1936
Conrad Wesselhoeft, Suffolk	June 2, 1937
Arrangements — Established 1849.	
G. Guy Bailey, Jr., Middlesex South	November 13, 1942 (appointed chairman May 19, 1947)
Franklin G. Balch, Jr., Suffolk	May 19, 1947

Gordon A. Donaldson, Middlesex South — May 19, 1947
Harold G. Giddings, Middlesex South — May 22, 1944
John W. Norcross, Middlesex South — May 19, 1947

Ethics and Discipline — Established 1871.

Ralph R. Stratton, Middlesex East — June 9, 1936 (appointed chairman May 21, 1941)
William J. Brickley, Suffolk — February 3, 1937
Archibald R. Gardner, Middlesex North — May 21, 1941
Fred R. Jouett, Middlesex South — May 21, 1940
Allen G. Rice, Hampden — June 1, 1938

Medical Education — Established 1881.

Chester S. Keefer, Suffolk — February 4, 1942 (appointed chairman May 19, 1947)
James M. Faulkner, Norfolk — May 21, 1946
George D. Henderson, Hampden — June 1, 1938
Isaac R. Jankelson, Norfolk — May 25, 1942

Membership — Established 1897.

Peirce H. Leavitt, Plymouth — June 1, 1938 (appointed chairman September 3, 1946)
William A. R. Chapin, Hampden — May 23, 1945
Lewis S. Pilcher, Middlesex South — July 26, 1946
Guy L. Richardson, Essex North — May 21, 1946
Samuel N. Vose, Suffolk — March 15, 1944
(Albert E. Parkhurst, Essex South; James M. Baty, Middlesex South; C. Bertram Gay, Worcester North; — representing the Supervising Censors)

Public Health — Established 1912.

Roy J. Ward, Worcester — May 22, 1944 (chairman)
John J. Poutas, Middlesex South — May 21, 1946
Warren R. Sisson, Suffolk — May 19, 1947
Lawrence J. Smith, Hampden — May 19, 1947
Conrad Wesselhoeft, Suffolk — July 27, 1944

Subcommittee of the Committee on Public Health

MENTAL HEALTH — Established 1947.

Walter E. Barton, Norfolk — February 5, 1947 (chairman)
William Malamud, Worcester — February 5, 1947
Henry A. Tadgell, Hampshire — February 5, 1947

Medical Defense — Established 1927.

Horatio Rogers, Suffolk — June 7, 1939 (appointed chairman May 19, 1947)
Edwin D. Gardner, Bristol South — June 7, 1927
Charles J. Kickham, Norfolk — May 21, 1946
John E. Moran, Franklin — May 19, 1947
William R. Morrison, Suffolk — June 9, 1936

Finance — Established 1938.

Robert W. Buck Middlesex South — May 21, 1946 (chairman)
Francis C. Hall, Suffolk — July 8, 1943
Fabyan Packard, Middlesex South — May 21, 1946
Bancroft C. Wheeler, Worcester — May 21, 1946
Charles F. Wilinsky, Suffolk — June 2, 1938

Society Headquarters — Established 1942.

Frank R. Ober, Suffolk — May 22, 1944 (appointed chairman November 1, 1944)
Albert A. Hornor, Suffolk — November 6, 1944
Walter G. Phippen, Essex South — May 21, 1946
Daniel B. Reardon, Norfolk South — May 22, 1944
George L. Steele, Hampden — May 19, 1947

Industrial Health — Established 1942.

Daniel L. Lynch, Norfolk — May 25, 1942 (appointed chairman May 21, 1946)
Joseph C. Aub, Suffolk — May 25, 1942
Louis R. Daniels, Middlesex South — May 22, 1944
John G. Downing, Middlesex South — May 22, 1944
Harold R. Kurth, Essex North — May 23, 1945
Frederic N. Manley, Norfolk South — May 19, 1947
Henry C. Marble, Suffolk — May 19, 1947

Advisory Committee to Committee on Industrial Health — Established 1942.

Philip Drinker
Harriet L. Hardy
Emma S. Tousant

SPECIAL COMMITTEES FOR 1947–1948

ELECTED BY THE COUNCIL, May 19, 1947

Cancer — Established 1917.

Shields Warren, Suffolk, *chairman*; Thomas J. Anglem, Suffolk; Ernest M. Daland, Suffolk; Allen G. Rice, Hampden; and Channing C. Simmons, Suffolk.

To Consider Expert Testimony — Established 1936.

Francis P. McCarthy, Norfolk, *chairman*; Carl Bearse, Norfolk; William J. Brickley, Suffolk; Frank R. Ober, Suffolk; and William H. Robey, Suffolk.

To Meet with the Massachusetts Hospital Association — Established 1940.

Albert E. Parkhurst, Essex South, *chairman*; Edward A. Adams, Worcester North; Edwin D. Gardner, Bristol South; Frederic Hagler, Hampden; Justin E. Hayes, Hampshire; and Nicholas S. Scarcello, Worcester.

Maternal Welfare — Established 1941.

Duncan Reid, Middlesex South, *chairman*; James M. Baty, Middlesex South; Arthur F. G. Edgelow, Hampden; Samuel B. Kirkwood, Middlesex East; Florence L. McKay, Suffolk; Louis E. Phaneuf, Suffolk; and Raymond S. Titus, Norfolk.

Rehabilitation — Established 1941.

Joseph H. Shortell, Suffolk, *chairman*; Benjamin F. Andrews, Worcester; Ralph M. Chambers, Bristol North; William M. Collins, Middlesex North; James J. Regan, Suffolk; and Arthur L. Watkins, Middlesex South.

Council Rules — Established 1944.

Charles E. Mongan, Middlesex South, *chairman*; Elmer S. Bagnall, Essex North; Frank R. Ober, Suffolk; George L. Schadt, Hampden; and Albert A. Hornor, Suffolk.

Postgraduate Medical Education — Established 1944.

W. Richard Ohler, Norfolk, *chairman*; George A. Buckley, Plymouth; Vlado A. Getting, Middlesex South; Robert H. Goodwin, Bristol South; Lewis M. Hurxthal, Suffolk; George P. Keaveny, Worcester North; Leo F. King, Middlesex North (interim appointment); Charles G. Mixter, Suffolk; Robert N. Nye, Suffolk; Joseph W. O'Connor, Worcester; Samuel H. Proger, Norfolk; George S. Reynolds, Berkshire; James L. Smead, Hampden; Harry C. Solomon, Suffolk; and Henry D. Stebbins, Essex South.

Medical Economics — Established 1944.

Leland S. McKittrick, Suffolk, *chairman*; Elmer S. Bagnall, Essex North; Allan M. Butler, Suffolk; Vlado A. Getting, Middlesex South; and Merrill C. Sosman, Suffolk.

Postwar Loan Fund — Established 1945.

George L. Schadt, Hampden, *chairman*; Eliot Hubbard, Jr., Middlesex South; Charles C. Lund, Suffolk; Stephen A. Mahoney, Hampden; and Albert E. Parkhurst, Essex South.

Physical Medicine — Established 1945.

Arthur L. Watkins, Middlesex South, *chairman*; Alexander P. Aitken, Middlesex East; Ralph M. Chambers, Bristol North; Franklin P. Lowry, Middlesex South; and Henry A. Tadgell, Hampshire.

To Make Recommendations as to Future Directors of Blue Shield — Established 1945.

Leland S. McKittrick, Suffolk, *chairman* (term expires 1949); Harold G. Giddings, Middlesex South (term expires 1951); Elliott P. Joslin, Suffolk (term expires 1952); Peirce H. Leavitt, Plymouth (term expires 1950); and George G. Smith, Suffolk (term expires 1948).

To Meet with General Hawley with View of Formulating Program in Massachusetts for Medical Care of Veterans and Dependents — Established 1945.

Humphrey L. McCarthy, Norfolk, *chairman*; James K. Bragger, Norfolk; and Allen S. Johnson, Hampden.

To Confer with the Massachusetts Farm Bureau Federation — Established 1945.

Joseph C. Merriam, Middlesex South, *chairman*; John E. Moran, Franklin; and Elmer E. Thomas, Hampshire.

To Make a Survey of Malpractice Insurance in Massachusetts — Established 1946.

Carl Bearse, Norfolk, *chairman*; William J. Brickley, Suffolk; Edwin D. Gardner, Bristol South; Daniel B. Reardon, Norfolk South; and Guy L. Richardson, Essex North.

To Assist the Council on Medical Education and Hospitals of the American Medical Association in the Provisional Approval of Certain Massachusetts Hospitals — Established 1946.

Robert T. Monroe, Norfolk, *chairman*; Laurence D. Chapin, Hampden; H. Quimby Gallupe, Middlesex South; Walter G. Phippen, Essex South; and Charles F. Wilinsky, Suffolk.

Veterans' Affairs — Established 1946.

G. Philip Grabfield, Suffolk, *chairman*; Victor G. Balboni, Suffolk; George P. Denny, Suffolk; Alexander Marble, Suffolk; and George F. Wilkins, Middlesex South.

In addition the following are representatives from the various district medical societies:

Charles H. Bradford, Suffolk
Stephen Brown, Hampshire
William M. Carr, Plymouth
Leo R. Desmond, Norfolk
Spencer C. Flo, Franklin
Merrill F. Gardner, Bristol South
Willis M. Gowen, Middlesex East
Leonard W. Hill, Bristol North
Sheldon L. Hunt, Barnstable
Thomas Hunter, Worcester
Edwin M. Mahoney, Hampden
John C. McGirr, Middlesex South
Frank P. Morse, Jr., Essex South
Franklin K. Paddock, Berkshire
Ensio K. F. Ronka, Norfolk South
Louis B. Simard, Essex North
Thomas J. G. Tighe, Middlesex North
Charles A. Wheeler, Worcester North

Postgraduate Assembly — Established 1946.

Leroy E. Parkins, Suffolk, *chairman*; Harold G. Giddings, Middlesex South; Frederick S. Hopkins, Hampden; Charles J. Kickham, Norfolk; and Robert N. Nye, Suffolk.

To Study Income Level for Blue Shield — Established 1946.

Charles F. Wilinsky, Suffolk, *chairman*; Norman H. Bruce, Middlesex South; Raoul L. Drapeau, Middlesex North; Henry L. Kirkendall, Worcester; and John W. Spellman, Norfolk.

To Establish a Pension Plan — Established 1947.

Robert W. Buck, Middlesex South, *chairman*; Eliot Hubbard, Jr., Middlesex South; Robert N. Nye, Suffolk; and George W. Papen, Norfolk.

Advisory Committee on School Medical Services — Established 1947.

Stewart Clifford, Middlesex South; Elmer S. Bagnall, Essex North; Joseph Garland, Suffolk; Florence McKay, Suffolk; Ernest Morris, Middlesex South; Thomas F. Reilly, Hampden; and James O. Wails, Worcester.

To Study Special Services — Established 1947.

Blue Cross: Elmer S. Bagnall, Mr. Reginald F. Cahalane.
Blue Shield: Arthur W. Allen, Charles G. Hayden.
Massachusetts Hospital Association: Mr. Frank Wing, Rev. Donald A. McGowan, Dr. Charles F. Wilinsky.
Massachusetts Medical Society: Leland S. McKittrick, *chairman*; Joseph Garland, *secretary*.
Radiologists: Merrill C. Sosman, Hugh F. Hare.
Pathologists: G. Kenneth Mallory, Donald A. Nickerson.
Anesthesiologists: Urban H. Eversole, Sidney C. Wiggin.

Auditing

Howard B. Jackson, Norfolk, *chairman*; and Frank T. Downey, Middlesex South.

Representatives to the Massachusetts Central Health Council.

Elmer S. Bagnall, Essex North, *chairman*; James W. Bunce, Berkshire; Merrill E. Champion, Suffolk; Earle M. Chapman, Suffolk.

Representative to the Hospital Council of Boston for the Year 1947.

William E. Browne, Suffolk.

Representative on the Legislative Committee of the Massachusetts Central Health Council.

John F. Conlin, Suffolk.

Representative on a Professional Advisory Committee Organized by the Division of Vocational Rehabilitation of the State Department of Education for the Purpose of Establishing a Program of Physical Restoration.

Joseph H. Shortell, Suffolk.

Representatives to the Council of the New England State Medical Societies.

Archibald J. Douglas, Hampden; Gerald N. Hoeffel, Middlesex South; and Norman A. Welch, Norfolk.

Representative for Survey by Academy of Pediatrics.

Gerald N. Hoeffel, Middlesex South.

Twenty-Five Voting Members in Massachusetts Hospital Service, Inc.

Benjamin H. Alton, Worcester; Gerardo M. Balboni, Suffolk; Laurence D. Chapin, Hampden; Lucien R. Chaput, Essex North; George K. Fenn, Essex South; Joseph E. Flynn, Middlesex South; Archibald R. Gardner, Middlesex North; Harold G. Giddings, Middlesex South; Henry W. Godfrey, Middlesex South; Albert A. Hornor, Suffolk; John H. Lambert, Middlesex North; Alexander A. Levi, Middlesex South; Raymond A. McCarthy, Middlesex South; Joseph C. Merriam, Middlesex South; Donald Munro, Suffolk; Albert E. Parkhurst, Essex South; Lewis S. Pilcher, Middlesex South; Helen S. Pittman, Suffolk; Allen G. Rice, Hampden; Arthur T. Ronan, Norfolk; George L. Steele, Hampden; Ralph R. Stratton, Middlesex East; John E. Talbot, Worcester; and Edward L. Young, Norfolk.

Delegates and Alternates to the House of Delegates of the American Medical Association for 1947–1948

Delegates	Alternates
June 1, 1946, to June 1, 1948	
David D. Scannell, Norfolk	Elmer S. Bagnall, Essex North
Dwight O'Hara, Middlesex South	William J. Pelletier, Franklin
Charles E. Mongan, Middlesex South	Patrick E. Gear, Hampden
Walter G. Phippen, Essex South	John I. B. Vail, Barnstable
June 1, 1947 to June 1, 1949	
Charles J. Kickham, Norfolk	John Fallon, Worcester
Leland S. McKittrick, Suffolk	Patrick J. Sullivan Berkshire

Councilors for 1947–1948

(Elected by the District Medical Societies at Their Annual Meetings, April 15 to May 15, 1946.)

Barnstable

H. P. Hopkins, Chatham, V. P.
P. M. Butterfield, Harwich, E. C., M. N. C.
P. P. Henson, Hyannis, 149 Main St., A. E. C., A. M. N. C., P. R. C.
J. G. Kelley, Pocasset, Barnstable County Sanatorium. Leg. C.
Frederick Sanborn, Osterville. Bates Ave., Sec.
John I. B. Vail, Hyannis, 155 Main St.

Berkshire

Modestino Criscitiello, Pittsfield, 28 North St., V. P.
D. N. Beers, Pittsfield, 74 North St., Sec.
N. N. Copeland, Pittsfield, 131 North St., A. M. N. C.
J. F. Flynn, Pittsfield, 28 North St.
John Hughes, Pittsfield, 74 North St., E. C., M. N. C., Leg. C.
C. T. Leslie, Pittsfield, 18 Bank Row.
N. B. McWilliams, Williamstown, 56 Spring St.
Helen M. Scoville, Pittsfield, House of Mercy Hospital.
P. J. Sullivan, Dalton, 471 Main St., P. R. C.
E. R. Wyman, Great Barrington, 259 Main St., A. E. C.

Bristol North

J. V. Chatigny, Taunton, 43 West Britannia St., V. P.
W. E. Dawson, Taunton, 58 Winthrop St., Sec.
M. E. Johnson, Attleboro, 33 Bank St., P. R. C.
C. B. Kingsbury, Taunton, 63 Prospect St., A. E. C., A. M. N. C.
J. L. Murphy, Taunton, 23 Cedar St., E. C., M. N. C.
W. M. Stobbs, Attleboro, 63 Bank St., Leg. C.

Bristol South

G. W. Blood, Fall River, 82 New Boston Rd., V. P.
R. B. Butler, Fall River, 278 No. Main St., E. C.
J. C. Corrigan, Fall River, 422 No. Main St.
J. E. Fell, Fall River. 181 Purchase St., Sec.
J. A. Fournier, Fall River, 11 Choate St.
E. D. Gardner, Marion, Box 175.
R. H. Goodwin, New Bedford, 15 South 6th St.
William Mason, Fall River, 151 Rock St.
D. R. Mills, Edgartown, Pease Point Way, A. M. N. C.
H. E. Perry, New Bedford, 159 Cottage St., P. R. C.
C. C. Tripp, New Bedford, 416 County St., A. E. C., Leg. C.
Henry Wardle, Fall River, 173 Purchase St., M. N. C.

Essex North

L. C. Peirce, Newburyport, 279 High St., V. P.
H. M. Allen, Lawrence, 301 Essex St.
E. S. Bagnall, Groveland, 281 Main St., Ex-Pres., C.
R. V. Baketel, Methuen, 7 Hampshire St.
G. J. Connor, Haverhill, 81 Merrimack St., A. E. C.
Elizabeth Councilman, Newburyport, 83 High St.
N. F. DeCesare, Lawrence, 57 Jackson St., Leg. C.
H. A. Fenton, Lawrence, 36 Winthrop Ave.
A. P. George, Haverhill, 78 Chestnut St.
H. R. Kurth, Lawrence, 57 Jackson St., Sec., P. R. C.
P. J. Look, Andover, 115 Main St., A. M. N. C.
R. C. Norris, Methuen, 247 Broadway, E. C.
G. L. Richardson, Haverhill, 94 Emerson St.
F. W. Snow, Newburyport, 24 Essex St.
C. F. Warren, Amesbury, 1 School St., M. N. C.

Essex South

L. F. Box, Beverly, 39 Broadway, V. P.
Bernard Appel, Lynn, 281 Ocean St., P. R. C., A. E. C.
W. W. Babson, Gloucester, 79 Prospect St.
K. J. Chadwell, Swampscott, 26 Lexington Circle.
R. E. Foss, Peabody, 125 Main St.
S. E. Goulding, Beverly, 38 Ocean St.
Loring Grimes, Swampscott, 84 Humphrey St., Leg. C.
C. A. Herrick, Manchester, 21 Union St.
W. R. Irving, Gloucester, 35 Middle St.
P. P. Johnson, Beverly, 1 Monument Square, M. N. C.
B. B. Mansfield, Ipswich, 4 Green St.
A. E. Parkhurst, Beverly, 1 Monument Square, C.
O. S. Pettingill, Middleton, Essex Sanatorium, A. M. N. C.
W. G. Phippen, Salem, 31 Chestnut St., Ex-Pres., E.C.
E. D. Reynolds, Danvers, 48 High St.
J. R. Shaughnessey, Salem, 24½ Winter St.
H. D. Stebbins, Salem, 342 Essex St., Sec.
P. E. Tivnan, Salem, 70 Washington St.
C. F. Twomey, East Lynn, 80 Ocean St.
C. A. Worthen, Lynn, 19 Park St.

Franklin

J. B. Temple, Shelburne Falls, 11 Main St., V. P.
H. L. Craft, Ashfield, Sec.
L. R. Dame, Greenfield, 78 Federal St., E. C.
F. W. Dean, East Northfield, 185 Main St., A. E. C.
H. M. Kemp, Greenfield, 42 Franklin St., P. R. C.
H. R. Mahar, Orange, 1 High St., Leg. C.
J. E. Moran, Greenfield, 31 Federal St., M. N. C.
W. D. Thomas, Montague, Central St., A. M. N. C.

Hampden

A. F. G. Edgelow, Springfield, 76 Maple St., V. P.
F. H. Allen, Holyoke, 16 Fairfield St.
E. P. Bagg, Holyoke, 207 Elm Street, President.
R. L. Barrett, Springfield, 21 Maple St.
H. F. Byrnes, Springfield, 6 Chestnut St.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Springfield, 333 Bridge St.
A. J. Douglas, Westfield, 30 Court St., E. C.
E. C. Dubois, Springfield, 174 Buckingham St.
P. E. Gear, Holyoke, 188 Chestnut St.
Frederic Hagler, Springfield, 20 Maple St., A. E. C., P. R. C.
G. D. Henderson, Holyoke, 176 Chestnut St.
F. S. Hopkins, Springfield, 146 Chestnut St.
Charles Jurist, Springfield, 70 Chestnut St.
R. T. Miller, Ware, 89 Main St.
John Pallo, Westfield, 97 Elm St.
A. G. Rice, Springfield, 146 Chestnut St.
A. H. Riordan, Indian Orchard, 147 Oak St., Leg. C.
G. L. Schadt, Springfield, 44 Chestnut St., Ex-Pres., M. N. C., C.
J. A. Seaman, Longmeadow. Office, Springfield, 20 Maple St.
G. C. Steele, West Springfield, 39 Upper Church St., Sec.
G. L. Steele, Springfield, 20 Maple St., A. M. N. C.
W. W. Teahan, Holyoke, 217 Essex St.

Hampshire

J. J. Curran, Northampton, 16 Centre St., V. P.
L. N. Durgin, Amherst, 66 Amity St., A. E. C., A. M. N. C., Leg. C.
J. R. Hobbs, Williamsburg, Main St., P. R. C.
L. B. Pond, Easthampton, 115 Main St.
F. Mary P. Snook. Worthington, Sec.
H. A. Tadgell, Belchertown, Belchertown State School, E. C., M. N. C.

Middlesex East

D. L. Joyce, Woburn, 269 Main St., V. P.
J. L. Anderson, Reading, 53 Woburn St., A. E. C.
T. P. Devlin, Stoneham, 38 Pleasant St.
Robert Dutton, Wakefield, 33 Avon St.
E. M. Halligan, Reading, 37 Salem St., M. N. C.
R. W. Layton, Melrose, 8 Porter St., Sec.
K. L. Maclachlan, Melrose 76, 1 Bellevue Ave., E. C.
M. J. Quinn, Winchester, 44 Church St., P. R. C.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, C.
J. M. Wilcox, Woburn, 6 Bennett St., Leg. C., A. M. N. C.

Middlesex North

C. S. Baker, Lowell, 8 Merrimack St., V. P.
R. E. Cole, Westford.
W. M. Collins, Lowell, 174 Central St., E. C., A. M. N. C.
S. A. Dibbins, Lowell, 528 Andover St., P. R. C.
L. J. Hall, Lowell, 8 Merrimack St.
B. D. Leahey, Lowell, 9 Central St., Sec.
W. F. Ryan, Lowell, 219 Central St., M. N. C.
A. W. Stearns, Billerica, Leg. C.
A. J. Stewart, Lowell, 310 Merrimack St., A. E. C.

Middlesex South

J. F. Casey, Allston. Office, Boston 15, 475 Commonwealth Ave., V. P.
G. G. Bailey, Jr., Boston, 412 Beacon St., C.
E. W. Barron, Malden 48. Office, Boston, 20 Ash St.
Harris Bass, Everett 49, 351 Broadway.
J. M. Baty, Belmont. Office, Brookline 46, 1101 Beacon St.
J. D. Bennett, West Somerville 44, 72 College Ave.
W. O. Blanchard, Newton 58, 465 Centre St.
H. K. Bloom, Everett, 17 Kenwood Rd.
G. F. H. Bowers, Newton Highlands 61, 156 Woodward St.
Alice M. Broadhurst, Watertown, 259 Mt. Auburn St.
Madelaine R. Brown, Cambridge. Office, Boston 16, 264 Beacon St.
R. N. Brown, Malden, 621 Main St.
R. W. Buck, Waban. Office, Boston 15, 5 Bay State Rd., C.
E. J. Butler, Cambridge, 25 Garden St.
C. W. Clark, Newtonville 60, 363 Walnut St.
E. A. Cooney, Newton. Office, Boston 16, 270 Commonwealth Ave.
W. H. Crosby, Brighton, 304 Faneuil St.
J. A. Daley, Natick, 36 Pond St.
C. L. Derick, Newton Highlands. Office, Boston 15, 412 Beacon St.
J. G. Downing, Newton. Office, Boston 15, 520 Commonwealth Ave.
A. G. Engelbach, Cambridge, 330 Mt. Auburn St.
W. C. Feeley, Cambridge, 859 Massachusetts Ave.
C. W. Finnerty, West Somerville 44, 440 Broadway.
H. Q. Gallupe, Waltham 54, 751 Main St.
F. W. Gay, Malden, 20 Park St.
V. A. Getting, Belmont. Office, Boston, 546 State House.
H. G. Giddings, Newton Centre. Office, Boston 16, 270 Commonwealth Ave., E. C., M. N. C.
H. W. Godfrey, Auburndale 66, 14 Hancock St.
J. L. Golden, Medford 55, 86 Forest St.
A. D. Guthrie, Medford 55, 408 Salem St.
Eliot Hubbard, Jr., Cambridge, 29 Highland St., Treasurer.
A. M. Jackson, Everett, 512 Broadway, A. E. C.
F. R. Jouett, Cambridge, 1 Craigie St., A. M. N. C.
Sylvester B. Kelley, West Newton, 34 Exeter St.
A. A. Levi, Newton. Office, Boston 15, 481 Beacon St., Sec.
A. N. Makechnie, Cambridge, 14 Upland Rd.
R. A. McCarty, Waltham 54, 465 Lexington St.
J. H. McSweeney, Somerville, 26 Bow St.
J. C. Merriam, Framingham, 198 Union Ave., C.
Dudley Merrill, Cambridge, 51 Brattle St.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres., C.
G. M. Morrison, Waban. Office, Boston 15, 520 Commonwealth Ave., C.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
E. J. O'Brien, Jr., Newton. Office, Boston 16, 270 Commonwealth Ave.
Dwight O'Hara, Waltham. Office, Boston 15, 416 Huntington Ave., Ex-Pres.
Fabyan Packard, Belmont, 154 Washington St.
L. G. Paul, Newton Centre. Office, Boston 16, 270 Commonwealth Ave.
L. S. Pilcher, Newton Centre 59, 43 Parker St.
Randolph Piper, Concord, 14 Sudbury Rd.
T. E. Reilly, Marlboro, 6 Newton St.
Max Ritvo, Newton. Office, Boston 15, 416 Marlboro St.
G. A. Saunders, Arlington, 50 Pleasant St.
M. J. Schlesinger, Newton. Office, Boston 15, 330 Brookline Ave.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
K. J. Tillotson, Waverley, McLean Hospital, Leg. C.
A. B. Toppan, Watertown, 289 Mt. Auburn St.
J. H. Townsend, Belmont, 195 Marsh St.
J. E. Vance, Natick. Office, Boston 15, 29 Bay State Rd.
C. F. Walcott, Cambridge, 81 Sparks St.
A. L. Watkins, Arlington. Office, Boston 14, Massachusetts General Hospital, C.
R. H. Wells, Lexington, 1430 Massachusetts Ave., P. R. C.
B. M. Wein, Newton. Office, Boston 15, 471 Commonwealth Ave.
B. S. Wood, Weston. Office, Waltham 54, 751 Main St.
Alfred Worcester, Waltham 54, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

Norfolk

Carl Bearse, Boston 15, 483 Beacon St., V. P., A. E. C., C.
A. A. Abrams, Brookline 46, 153 Dean Rd.
C. E. Allard, Dorchester, 428 Columbia Rd., A. M. N. C.
G. R. Allen, Norwood, 449 Washington St.
B. E. Barton, West Roxbury 32, 10 Richwood St., Sec.
Elizabeth Broyles, Wellesley, Simpson Infirmary.
J. H. Cauley, Dorchester, 8 Carruth St.
L. R. Desmond, Milton, 1272 Brook Rd.
G. L. Doherty, West Roxbury. Office, Boston 15, 466 Commonwealth Ave.
Albert Ehrenfried, Brookline. Office, Boston 15, 520 Beacon St., M. N. C.
J. M. Faulkner, Brookline. Office, 80 East Concord St., Boston.
Susannah Friedman, Roxbury. Office, Boston 15, 485 Commonwealth Ave.
T. R. Goethals, Brookline, 34 Hawthorn Rd.
D. L. Halbersleben, Brookline 46, 42 Goodnough Rd.
J. B. Hall, Roxbury 19, 108 Dudley St.
H. B. Harris, East Milton. Office, Dorchester, 487 Columbia Rd.
R. J. Heffernan, Jamaica Plain. Office, Brookline, 1101 Beacon St.
Gilbert Horrax, Brookline. Office, Boston, 605 Commonwealth Ave.
P. J. Jakmauh, Milton. Office, South Boston 27, 509 Broadway.
I. R. Jankelson, Jamaica Plain. Office, Boston 15, 483 Beacon St.
C. J. Kickham, Brookline. Office, Boston 15, 508 Commonwealth Ave., Vice-Pres.
C. J. E. Kickham, Jamaica Plain. Office, Brookline, 1101 Beacon St., E. C.
D. L. Lionberger, Dedham, 709 East St.
D. S. Luce, Canton, 553 Washington St., P. R. C.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale. Office, Boston, 245 State St., C.
T. F. P. Lyons, Milton. Office, Boston 16, 270 Commonwealth Ave.
F. P. McCarthy, Milton. Office, Boston 15, 371 Commonwealth Ave.
H. L. McCarthy, West Roxbury. Office, Boston 15, 479 Beacon St., C.
R. T. Monroe, Brookline. Office, Boston 16, 270 Commonwealth Ave., C.
F. J. Moran, Dedham, 395 Washington St.
H. R. Morrison, Wellesley. Office, Boston, 370 Marlboro St.
Hyman Morrison, Brookline. Office, Boston 15, 483 Beacon St.
D. J. Mullane, Brookline 46, 1101 Beacon St.
H. A. Novack, Brookline. Office, Boston, 471 Commonwealth Ave.
J. J. O'Connell, Dorchester, 1061 Dorchester Ave.
W. R. Ohler, Jamaica Plain. Office, Boston 15, 319 Longwood Ave., C.
E. E. O'Neil, Brookline. Office, Boston, 270 Commonwealth Ave.
G. W. Papen, Brookline. Office, Boston, 31 Milk St.
H. C. Petterson, West Roxbury. Office, Boston 15, 29 Bay State Rd.
S. H. Proger, Brookline. Office, Boston, 30 Bennet St.
Frederick Reis, Jamaica Plain. Office, Boston 15, 416 Huntington Ave.
H. A. Rice, Canton, 742 Washington St.

S. A. Robins, Boston 15, 636 Beacon St.
D. D. Scannell, Jamaica Plain. Office, Boston 15, 475 Commonwealth Ave.
J. A. Seth, Milton. Office, Boston 15, 47 Bay State Rd.
J. A. Sieracki, Norwood, 71 Winter St.
S. L. Skvirsky, Chestnut Hill. Office, Boston, 336 State House, Leg. C.
E. C. Smith, Brookline. Office, Boston 15, 520 Commonwealth Ave.
Kathleyne S. Snow, Jamaica Plain. Office, Boston 15, 466 Commonwealth Ave.
J. W. Spellman, Chestnut Hill. Office, Brookline, 1101 Beacon St.
A. R. Stagg, Medfield, 25 Pleasant St.
W. J. Walton, Dorchester, 106 Bowdoin St.
N. A. Welch, West Roxbury. Office, Boston 15, 520 Commonwealth Ave., Asst. Treas., C.
P. R. Withington, Milton, 350 Randolph Ave.
Marjorie Woodman, Jamaica Plain. Office, Boston, 21 Bay State Rd.
E. T. Wyman, Brookline. Office, Boston 15, 319 Longwood Ave.

Norfolk South

R. L. Cook, Quincy, 1245 Hancock St., V. P.
F. A. Bartlett, Wollaston 70, 308 Beale St., E. C.
D. L. Belding, Hingham, 215 Main St., Leg. C.
Harry Braverman, Quincy 69, 43 School St., A. E. C.
E. M. Britton, Wollaston, 25 Elm Ave.
W. R. Helfrich, Quincy 69, 166 Washington St.
Frederick Hinchliffe, Cohasset 117 South Main St., A. M. N. C.
E. K. Jenkins, South Braintree, Norfolk County Hospital, Sec.
N. R. Pillsbury, South Braintree, Norfolk County Hospital.
D. B. Reardon, Quincy 69, 1186 Hancock St., President-Elect.
H. A. Robinson, Hingham, 205 North St., P. R. C., M. N. C.
R. G. Vinal, Norwell, Main St.

Plymouth

R. C. McLeod, Brockton, Goddard Hospital, V. P.
J. C. Angley, Bryantville, School St., A. E. C.
A. L. Duncombe, Brockton, 38 Winthrop St., A. M. N. C., Leg. C.
Samuel Gale, Brockton, The Checkerton, Sec.
W. C. Gould, Kingston, 214 Main St.
P. H. Leavitt, Brockton, 129 West Elm St., C.
C. D. McCann, Brockton, 12 Cottage St., P. R. C.
G. A. Moore, Brockton, 167 Newbury St., E. C.
B. H. Peirce, South Hanson, Plymouth County Hospital, M. N. C.
E. L. Perry, Middleboro, 39 Oak St.

Suffolk

C. C. Lund, Boston 15, 20 Gloucester St., V. P.
H. L. Albright, Boston 15, 412 Beacon St.
T. J. Anglem, Brookline, 1180 Beacon St.
C. H. Bradford, Boston, 220 Beacon St.
W. J. Brickley, Boston 15, 524 Commonwealth Ave.
W. E. Browne, Boston 15, 587 Beacon St., Leg. C.
A. M. Butler, Boston 14, Massachusetts General Hospital.
A. J. A. Campbell, Boston 15, 520 Commonwealth Ave., E. C.
E. M. Chapman, Brookline. Office, Boston, 266 Beacon St.
Henry Clifford, Cambridge, 21 Lowell St.
A. P. DerHagopian, Chelsea, 39 Cary Ave.
H. H. Faxon, Brookline. Office, Boston, 264 Beacon St.
N. W. Faxon, Boston 14, Massachusetts General Hospital.
Jacob Fine, Boston 15, 330 Brookline Ave.
Maurice Fremont-Smith, Boston 15, 12 Hereford St.
Joseph Garland, Boston 16, 266 Beacon St., Secretary.
G. Lynde Gately, East Boston, 624 Bennington St.
A. A. Hornor, Boston 15, 319 Longwood Ave., M. N. C.
C. S. Keefer, Boston, 65 East Newton St., C.
H. A. Kelly, Winthrop, 200 Pleasant St.
T. H. Lanman, Boston 15, 300 Longwood Ave.
*C. F. Maraldi, Boston, 276 Commonwealth Ave.
F. W. Marlow, Jr., Brookline, 1284 Beacon St.
Donald Munro, Boston 18, 818 Harrison Ave.
H. L. Musgrave, Revere, 620 Beach St.
R. N. Nye, Boston 15, 8 Fenway.
F. R. Ober, Boston, 234 Marlboro St., C.
F. W. O'Brien, Boston 15, 475 Beacon St.
J. P. O'Hare, Boston 15, 520 Commonwealth Ave.
L. E. Parkins, Boston 15, 12 Bay State Rd., C.
L. E. Phaneuf, Boston 16, 270 Commonwealth Ave.
Helen S. Pittman, Boston 16, 264 Beacon St.
J. H. Pratt, Boston 11, 30 Bennet St.
J. J. Regan, South Boston. Office, Boston, 520 Commonwealth Ave.
Horatio Rogers, Boston, 264 Beacon St., C.
H. F. Root, Boston 15, 81 Bay State Rd., A. E. C., P. R. C.
Wyman Richardson, Newton Centre. Office, Boston, 264 Beacon St.
C. G. Shedd, Boston, 422 Beacon St., Sec.
R. M. Smith, Boston, 33 Dartmouth St., C.
C. M. Stearns, Chelsea, 116 Hawthorn St.
Augustus Thorndike, Boston 15, 319 Longwood Ave.
Conrad Wesselhoeft, Boston, 315 Marlboro St., A. M. N. C.

Worcester

F. B. Carr, Worcester, 27 Elm St., V. P.
A. W. Atwood, Worcester, 390 Main St.
George Ballantyne, Worcester, 27 Elm St.
F. P. Bousquet, Worcester, 390 Main St., A. E. C., A. M. N. C.
J. J. Cohen, Worcester, 340 Main St.
E. J. Crane, Holden, Armington Lane.
Paul Dufault, Rutland, Rutland State Sanatorium.
G. R. Dunlop, Worcester, 53 Massachusetts Ave., Leg. C.
W. J. Elliott, Worcester, 119 Belmont St.
John Fallon, Worcester, 390 Main St.
L. M. Felton, Worcester, 36 Pleasant St.
R. H. Goodale, Worcester, 36 Otsego Rd.
Thomas Hunter, Shrewsbury, 545 Main St., C.
H. L. Kirkendall, Worcester, 27 Elm St.
J. A. Lundy, Oxford, 26 Main St.
D. K. McClusky, Worcester, 7 Hawthorne St.
J. M. Olson, Westboro, 54 West Main St.
F. A. O'Toole, Clinton, 181 Chestnut St.
R. S. Perkins, Worcester, 27 Elm St., M. N. C.
E. L. Richmond, Worcester, 390 Main St.
N. S. Scarcello, Worcester, 1 Shelden St., P. R. C.
R. F. Sullivan, Worcester, 54 Hillcroft Ave.
J. J. Tegelberg, Worcester, 390 Main St., Sec.
G. C. Tully, Worcester, 1 Cedar St.
R. J. Ward, Worcester, 9 Bellevue St., C.
B. C. Wheeler, Worcester, 27 Elm St., E. C.

Worcester North

J. C. Hales, Gardner, 183 Lawrence St., V. P.
J. J. Curley, Leominster, 89 West St., E. C., M. N. C., Leg. C.
C. B. Gay, Fitchburg, 62 Day St.
G. P. Keaveny, Fitchburg, 62 Fox St., A. E. C.
C. N. McPeak, Fitchburg, 18 Hartwell St.
J. V. McHugh, Leominster, 55 West St., A. M. N. C., P. R. C.
J. G. Simmons, Fitchburg, 30 Myrtle Ave., Sec.

The initials *E.C.* following the name of a Councilor indicate that he is a member of the Executive Committee, and *A.E.C.* that he is an alternate member of the Executive Committee; *M.N.C.* that he is a member of the Committee on Nominations and *A.M.N.C.* that he is an alternate member of the Committee on Nominations; *Leg. C.* that he is a member of the Committee on Legislation; *P.R.C.* that he is a member of the Committee on Public Relations; *V.P.* that a member is a councilor by virtue of his office as president of a district society and so vice-president of the general society; *C.* by virtue of his office as chairman of a standing committee; *Sec.* by virtue of his office as secretary of a district society and *Ex-Pres.* by virtue of being a past president.

*Interim appointment.

Censors for 1947–1948

Barnstable
P. P. Henson, Hyannis, *supervisor.*
D. H. Hiebert, Provincetown.
D. E. Higgins, Barnstable.
Joseph Kelley, Orleans.
O. S. Simpson, Barnstable.

Berkshire
C. T. Leslie, Pittsfield, *supervisor.*
I. S. F. Dodd, Pittsfield.
A. C. England, Pittsfield.
W. T. Frawley, Pittsfield.
A. M. Gangemi, North Adams.

Bristol North
J. L. Murphy, Taunton, *supervisor.*
J. H. Brewster, Attleboro.
C. B. Kingsbury, Taunton.
A. J. Leddy, Taunton.
H. G. Vaughan, Attleboro.

Bristol South
Henry Wardle, Fall River, *supervisor.*
F. M. Howes, New Bedford.
W. F. MacKnight, Fall River.
E. A. McCarthy, Fall River.
C. C. Persons, New Bedford.

Essex North
R. V. Baketel, Methuen, *supervisor.*
M. F. Ames, Newburyport.
F. C. Atkinson, North Andover.
L. C. Peirce, Newburyport.
L. B. Simard, Haverhill.

Essex South
A. E. Parkhurst, Beverly, *supervisor.*
H. N. Baker, Rockport.
S. N. Gardner, Salem.
W. C. Inman, Danvers.
C. F. Twomey, Lynn.

Franklin
J. E. Moran, Greenfield, *supervisor.*
J. P. Colleran, South Deerfield.
F. W. Dean, Northfield.
H. R. Mahar, Orange.
W. J. Pelletier, Turners Falls.

Hampden
A. A. Palermo, Springfield, *supervisor.*
J. M. Gilchrist, Springfield.
G. D. Henderson, Holyoke.
John Pallo, Westfield.
J. L. Smead, Springfield.

Hampshire
L. B. Pond, Easthampton, *supervisor.*
Stephen Brown, Northampton.
M. E. Cooney, Northampton.
T. F. Corriden, Northampton.
J. E. Hayes, Northampton.

Middlesex East
S. H. Moses, Winchester, *supervisor.*
C. R. Baisley, Reading.
H. A. Bouve, Wakefield.
T. P. Devlin, Stoneham.
J. H. Fay, Melrose.

Middlesex North
W. F. Ryan, Lowell, *supervisor.*
L. F. King, Lowell.
E. H. Latham, Lowell.
H. L. Leland, Lowell.
J. D. Sweeney, Lowell.

Middlesex South
J. M. Baty, Belmont, *supervisor.*
Oliver Cope, Cambridge.
H. J. Crumb, Lexington.
E. A. Gaston, Framingham.
J. F. Williams, Everett.

Norfolk
Kathleyne S. Snow, Jamaica Plain, *supervisor.*
C. E. Allard, Dorchester.
I. R. Jankelson, Jamaica Plain.
H. A. Novack, Brookline.
E. E. O'Neil, Chestnut Hill.

Norfolk South
F. A. Bartlett, Wollaston, *supervisor.*
Arthur Rappeport, Quincy.
H. S. Reid, Cohasset.
R. E. Ross, South Braintree.
W. L. Sargent, Quincy.

Plymouth
E. L. Perry, Middleboro, *supervisor.*
L. A. Alley, Lakeville.
Jacob Brenner, North Easton.
F. B. Gilmore, Brockton.
R. E. Swenson, Plymouth.

Suffolk
J. H. Pratt, Boston, *supervisor.*
John F. Collins, Revere.
R. L. Goodale, Boston.
L. M. Hurxthal, Boston.
J. J. Todd, Boston.

Worcester
B. C. Wheeler, Worcester, *supervisor.*
J. B. Butts, Worcester.
E. J. Crane, Holden.
H. L. Kirkendall, Worcester.
J. W. McKoan, Jr., Worcester.

Worcester North
C. B. Gay, Fitchburg, *supervisor.*
K. J. Jolma, Gardner.
J. W. Mason, Ashburnham.
S. I. Nathanson, Fitchburg.
A. B. Skelton, Winchendon.

Vice-Presidents of the Massachusetts Medical Society (*Ex-Officiis*) for 1947–1948

Presidents of District Medical Societies

(Arranged according to seniority of fellowship in the Massachusetts Medical Society)

Bristol South — George W. Blood, Fall River.
Middlesex North — C. Stoyle Baker, Lowell.
Middlesex South — John F. Casey, Boston.
Norfolk — Carl Bearse, Boston.
Suffolk — Charles C. Lund, Boston.
Hampden — Arthur F. G. Edgelow, Springfield.
Middlesex East — Daniel L. Joyce, Woburn.
Bristol North — Joseph V. Chatigny, Taunton.
Berkshire — Modestino Criscitiello, Pittsfield.
Worcester North — Jesse C. Hales, Gardner.
Plymouth — Ralph C. McLeod, Plymouth.
Norfolk South — Robert L. Cook, Quincy.
Essex North — Lincoln C. Peirce, Newburyport.
Essex South — Leonard F. Box, Beverly.
Worcester — Frank B. Carr, Worcester.
Franklin — John B. Temple, Shelburne Falls.
Hampshire — John J. Curran, Northampton.
Barnstable — Henry P. Hopkins, Chatham.

Commissioners of Trial for 1947–1948

Barnstable — F. O. Cass, Provincetown.
Berkshire — J. B. Thomes, Pittsfield.
Bristol North — J. W. Cook, Mansfield.
Bristol South — A. C. Lewis, Fall River.
Essex North — F. W. Anthony, Haverhill.
Essex South — O. C. Blair, Lynn.
Franklin — K. W. D. Jacobus, Turners Falls.
Hampden — F. K. Dutton, Springfield.
Hampshire — R. C. Byrne, Hatfield.
Middlesex East — I. W. Richardson, Wakefield.
Middlesex North — C. M. Roughan, Lowell.
Middlesex South — H. P. Stevens, Cambridge.
Norfolk — W. J. Walton, Dorchester.
Norfolk South — W. L. Sargent, Quincy.
Plymouth — J. A. Carriulo, Brockton.
Suffolk — D. G. Anderson, Boston.
Worcester — W. P. Bowers, Clinton.
Worcester North — W. E. Currier, Leominster.

Officers of the Sections for 1947–1948

Medicine

Chairman, Francis C. Hall, Boston; *vice-chairman*, Laurence B. Ellis, Boston; *secretary*, Allen S. Johnson, Springfield.

Surgery

Chairman, George S. Reynolds, Pittsfield; *secretary*, Robert E. Gross, Boston.
Executive Committee — John W. Spellman, Brookline; Charles C. Lund, Boston; Langdon Parsons, Newton.

Pediatrics

President, Lewis W. Hill, Boston; *secretary*, Gerald N. Hoeffel, Boston and Cambridge.
Executive Committee — Leroy T. Stokes, Haverhill; Floyd R. Smith, Pittsfield; Hyman Green, Boston.

Obstetrics and Gynecology

Chairman, Arthur T. Hertig, Boston; *vice-chairman*, James F. Conway, Brookline; *secretary*, Daniel J. McSweeney, Milton.

Radiology

Chairman, Hugh F. Hare, Newton and Boston; *secretary*, Albert M. Moloney, West Roxbury and Boston.

Physiotherapy

Chairman, William D. McFee, Haverhill; *secretary*, Henry A. Tadgell, Belchertown.

Dermatology and Syphilology

Chairman, Austin W. Cheever, Boston; *secretary*, Maurice M. Tolman, Chelsea.

Anesthesiology

Chairman, Urban H. Eversole, Boston; *secretary*, Morris J. Nicholson, Boston.

Officers of the District Medical Societies for 1947–1948

Barnstable — *President*, Henry P. Hopkins, Chatham; *vice-president*, Arthur J. D'Elia, Harwich Port; *secretary*, Frederick Sanborn, Barnstable; *treasurer*, Frank Travers, Barnstable; *librarian*, Carroll H. Keane, Chatham; *executive councilor* and *public relations councilor*, Paul P. Henson, Hyannis.

Berkshire — *President*, Modestino Criscitiello, Pittsfield; *vice-president*, George S. Reynolds, Pittsfield; *secretary*, Daniel N. Beers, Pittsfield; *treasurer*, Theodore W. Jones, Pittsfield; *legislative councilor*, John Hughes, Pittsfield; *public relations councilor*, Patrick J. Sullivan, Dalton.

Bristol North — *President*, Joseph V. Chatigny, Taunton; *vice-president*, James H. Brewster, Attleboro; *secretary*, William E. Dawson, Taunton; *treasurer*, Charles E. Hoye, Taunton; *executive councilor*, J. L. Murphy, Taunton; *legislative councilor*, William M. Stobbs, Attleboro; *public relations councilor*, M. E. Johnson, Attleboro.

Bristol South — *President*, George W. Blood, Fall River; *vice-president*, Aubrey J. Pothier, New Bedford; *secretary* and *treasurer*, James E. Fell, Fall River; *executive councilor*, Richard B. Butler, Fall River; *legislative councilor*, Curtis C. Tripp, New Bedford; *public relations councilor*, Harold E. Perry, New Bedford.

Essex North — *President*, Lincoln C. Peirce, Newburyport; *vice-president*, Robert E. Blais, Amesbury; *secretary*, Harold R. Kurth, Methuen; *treasurer*, J. Leroy Wood, Methuen; *executive councilor*, Rolf C. Norris, Methuen; *legislative councilor*, Nicandro F. DeCesare, Methuen; *public relations councilor*, Harold R. Kurth, Methuen.

Essex South — *President*, Leonard F. Box, Beverly; *vice-president*, Alexander Kotarski, Peabody; *secretary*, Henry D. Stebbins, Marblehead; *treasurer*, Andrew Nichols, III, Danvers; *legislative councilor*, Loring Grimes, Swampscott; *public relations councilor*, Bernard Appel, Lynn; *executive councilor*, Walter G. Phippen, Salem.

Franklin — *President*, John B. Temple, Shelburne Falls; *vice-president*, Lawrence R. Dame, Greenfield; *secretary* and *treasurer*, Harry L. Craft, Ashfield; *public relations councilor*, Howard M. Kemp, Greenfield.

Hampden — *President*, Arthur F. G. Edgelow, Springfield; *vice-president*, Robert Hildreth, Westfield; *secretary* and *treasurer*, George C. Steele, West Springfield; *executive councilor*, Archibald J. Douglas, Westfield; *legislative councilor*, Arthur Riordan, Indian Orchard; *public relations councilor*, Frederic Hagler, Springfield.

Hampshire — *President*, John J. Curran, Northampton; *vice-president*, Edward J. Manwell, Northampton; *secretary* and *treasurer*, F. Mary P. Snook, Worthington; *librarian*, Abbie M. O'Keefe, Northampton; *executive councilor*, Henry A. Tadgell, Belchertown; *legislative councilor*, Lawrence N. Durgin, Amherst; *public relations councilor*, Joseph R. Hobbs, Williamsburg.

Middlesex East — *President*, Daniel L. Joyce, Woburn; *vice-president*, Milton J. Quinn, Winchester; *secretary*, Roy W. Layton, Melrose; *treasurer*, Albert E. Small, Winchester; *librarian*, Angelo L. Maietta, Winchester; *public relations councilor*, Milton J. Quinn, Winchester.

Middlesex North — *President*, C. Stoyle Baker, Lowell; *vice-president*, James Y. Rodger, Lowell; *secretary*, Brendan D. Leahey, Lowell; *treasurer*, Mason D. Bryant, Lowell; *executive councilor*, William M. Collins, Lowell; *legislative councilor*, A. Warren Stearns, Lowell; *public relations councilor*, Samuel Dibbins, Lowell.

Middlesex South — *President*, John F. Casey, Allston; *vice-president*, Arthur M. Jackson, Everett; *secretary*, Alexander A. Levi, Newton; *treasurer*, Fabyan Packard, Belmont; *orator*, Vlado A. Getting, Belmont; *legislative councilor*, Kenneth J. Tillotson, Belmont; *public relations councilor*, Ralph H. Wells, Lexington; *executive councilor*, Harold G. Giddings, Newton.

Norfolk — *President*, Carl Bearse, Newton; *vice-president*, George W. Papen, Brookline; *secretary*, Basil E. Barton, West Roxbury; *treasurer*, Frederick Reis, Jamaica Plain; *executive councilor*, Charles J. E. Kickham, Jamaica Plain; *legislative councilor*, Solomon L. Skvirsky, Chestnut Hill; *public relations councilor*, Dean S. Luce, Canton.

Norfolk South — *President*, Robert L. Cook, Quincy; *vice-president*, George D. Dalton, Wollaston; *secretary*, Ebenezer K. Jenkins, Braintree; *treasurer*, Francis G. King, North Quincy; *librarian*, Ebenezer K. Jenkins, Braintree; *public relations councilor*, Henry A. Robinson, Hingham; *executive councilor*, Fred A. Bartlett, Hingham; *legislative councilor*, David L. Belding, Hingham.

Plymouth — *President*, Ralph C. McLeod, Brockton; *vice-president*, Sterling A. McLean, Middleboro; *secretary*, Samuel Gale, Brockton; *treasurer*, Alton L. Hurlburt, East Bridgewater; *librarian*, John H. Weller, Bridgewater; *executive councilor*, George A. Moore, Brockton; *public relations councilor*, Charles D. McCann, Brockton; *legislative councilor*, Alfred L. Duncombe, Brockton.

Suffolk — *President*, Charles C. Lund, Boston; *vice-president*, Hollis L. Albright, Boston; *secretary*, Charles G. Shedd, Boston; *treasurer*, Richard S. Eustis, Boston; *executive councilor*, Alexander J. A. Campbell, Boston; *legislative councilor*, William E. Browne, Boston; *public relations councilor*, Howard F. Root, Boston.

Worcester — *President*, Frank B. Carr, Worcester; *vice-president*, John J. Dumphy, Worcester; *secretary*, Julius J. Tegelberg, Worcester; *treasurer*, Arthur D. Ward, Worcester; *librarian*, John Fallon, Worcester; *executive councilor*, Bancroft C. Wheeler, Worcester; *legislative councilor*, George R. Dunlop, Worcester; *public relations councilor*, Nicholas S. Scarcello, Worcester.

Worcester North — *President*, Jesse C. Hales, Gardner; *vice-president*, Donald B. Cheetham, Athol; *secretary*, James G. Simmons, Fitchburg; *treasurer*, Frederick H. Thompson, Jr., Fitchburg; *executive and legislative councilor*, John J. Curley, Leominster; *public relations councilor*, James V. McHugh, Leominster.

Admissions Recorded from May 22, 1946 to May 22, 1947

Year of Admission	Name and Residence	Medical School
1946	Abele, Virgil, Framingham	Middlesex
1946	Abriel, Albert G., Medford Hillside	Boston University
1946	Adams, Crawford W., Malden	Boston University
1946	Adams, Lambi N., Worcester	Hahnemann Medical College
1947	Adler, Morris H., Newton	University of Vienna
1946	*Adzigian, Nazareth, Newton Lower Falls	Tufts
1947	Albano, Peter R., Great Barrington	Middlesex
1946	Albert, Harold S., Boston	McGill University
1946	Alexander, Eben, Jr., Brookline	Harvard
1947	Alexander, Fred, Boston	University of Maryland
1946	Alford, Hyman, Dorchester	Tufts
1946	Amerena, John P., Randolph	Middlesex
1946	*Anderson, Albert B., Cambridge	Harvard
1947	Anton, Harry J., Three Rivers	University of Vermont
1947	Appel, John F., Holyoke	Jefferson Medical College
1946	Arnone, William H., Brockton	New York University
1947	Arthurs, Alexander T., Somerville	Middlesex
1946	*Ayres, John C., Brighton	Boston University
1946	Bailey, David B., Lowell	Tufts
1947	Baker, Donald V., Uxbridge	Harvard
1947	Baldry, George S., Quincy	University of Manitoba
1946	Ballin, Ludwig, South Braintree	University of Munich
1946	Bartol, George, Wellesley Hills	University of Pennsylvania
1947	Barton, David J., Everett	Johns Hopkins University
1946	*Bassett, Gardner G., Brockton	Harvard
1947	Bautze, Frank A., Baldwinsville	Harvard
1946	Baver, Charles L., Stockbridge	Middlesex
1947	Beakey, John F., Cambridge	Tufts
1946	Bedrick, Morton, Fall River	Harvard
1947	Beer, Freda S., Waltham	Friedrich–Wilhelm University
1946	Benoit, Noe N., Millbury	Tufts
1946	Berk, Morton S., Newton Centre	Boston University
1947	Berry, Francis D., Milford	Tufts
1947	Bicchieri, N. Anthony, Roslindale	University of London
1947	Binder, Abraham A., Brookline	Boston University
1947	Blechman, Benjamin, Lowell	Leipzig University
1946	Bluestein, Louis L., Hyde Park	Middlesex
1947	Blute, James F., Jr., Watertown	Harvard
1947	Boardman, Donnell W., Acton	Columbia University
1947	Bolino, Armand V., Lynn	Kansas City University of Physicians and Surgeons
1947	Bourne, George C., Hyannis	Tufts
1946	Bragdon, Joseph H., Dedham	Columbia University
1946	Breed, Frederic B., Gloucester	Harvard
1947	*Bresnick, Elliott, Brookline	Tufts
1947	Brewster, Henry H., Milton	Harvard
1947	Bridges, William C., Newton Centre	Yale
1946	Briggs, Bernard D., Stoneham	College of Medical Evangelists
1947	Brochu, Charles E., Webster	Georgetown University
1946	Brooks, Eugene F., Wrentham	Middlesex
1947	Brougham, Milton F., Wollaston	Harvard
1946	Brown, Chester W., Worcester	University of Rochester
1946	Brown, Harold F., Wellesley Hills	Tufts
1947	Brown, Robert H., Foxboro	Harvard
1946	Bruno, Salvatore J., Medford	New York Medical College
1947	Bryan, Burton D., Fall River	University of Vermont
1946	Buckley, William R., Medford	Boston University
1946	Burden, Charles N., Taunton	Tufts
1947	Burger, Harold, New Bedford	Boston University
1947	Burnett, Charles H., Newtonville	University of Colorado
1946	*Bush, James F., Monson	Tufts
1946	*Butman, Douglas E., Waltham	Harvard
1947	Byrne, John J., Weston	Harvard
1947	Cahan, Alvin M., Boston	Cornell University
1947	Campbell, Elmore M., Dorchester	Tufts

1947	Campbell, James B., Jamaica Plain	Harvard
1946	Capodilupo, Graziano A., Boston	Middlesex
1946	Caradonna, Matteo, Everett	Middlesex
1947	Cardillo, Edward M., Everett	Middlesex
1946	*Carr, Francis J., Somerville	Boston University
1946	Carroll, John J., Dedham	Tufts
1946	Carter, Barbard F., Boston	Tufts
1946	*Carter, Sidney, Reading	Boston University
1946	Cassel, Arthur L., Great Barrington	University of Rostock
1946	Cavanaugh, Thomas E., Jr., West Springfield	Georgetown University
1946	Ceplikas, Thomas A., North Andover	Tufts
1947	Chalmers, Thomas C., Jr., Cambridge	Columbia University
1947	Chasen, William H., Roslindale	University of Lausanne
1947	Cheney, Roger H., Springfield	University of Pennsylvania
1946	*Chretien, Thomas E., Auburndale	Tufts
1947	Clancy, George F., Marlboro	Georgetown University
1947	Cleary, Robert V.. Cambridge	Harvard
1946	Clinton, Marshall, Jr., Wellesley	University of Buffalo
1947	Coburn, Morton B., New York	Middlesex
1946	Cohen, Sidney, Boston	Harvard
1946	Collins, Gerald M., Hingham	Boston University
1946	*Colozzi, Anthony E., Lexington	Boston University
1947	Conlan, William P., Holbrook	Boston University
1947	Connors, Raymond J., Fall River	Harvard
1946	*Covell, Lester L., Woburn	Tufts
1946	Curley, George A., Mattapan	College of Physicians and Surgeons, Boston
1947	Daiell, Saul S., West Roxbury	Louisiana State University
1947	Daly, Bernard J., Lawrence	Georgetown University
1946	*Dana, Jacob B., Brighton	Boston University
1947	D'Avanzo, Charles S., West Springfield	New York Medical College
1946	David, Lloyd G., Lowell	Tufts
1947	Davis, Jean P., Wellesley	Yale
1947	Deacon, Walter E., South Duxbury	Tufts
1946	Dean, Michael A., Millville	Tufts
1946	Deering, George E., Worcester	Harvard
1946	Dennehy, Timothy J., Chestnut Hill	Georgetown University
1946	Diamand, Morris, Athol	University of Vienna
1947	Diamond, Charles A., West Springfield	New York Medical College
1946	Dine, Robert F., Brookline	Yale
1946	Donoghue, William F., Jr., Springfield	Tufts
1946	Doret, Stanley A., Greenfield	Tufts
1946	*Dorgan, Joseph A., Brighton	Tufts
1946	Dorman, Daniel B., Pittsfield	Harvard
1947	Dove, David, Beverly	Harvard
1947	Driscoll, Cornelius J., Danvers	Georgetown University
1946	Ehrenberg, Ruth, Dorchester	University of Berlin
1947	Eichwald, Ernst J., North Weymouth	Albert-Ludwig University
1946	*Emerson, George F., South Weymouth	Harvard
1946	Emerson, Kendall, Jr., Brookline	Harvard
1946	England, Albert C., Jr., Brookline	Harvard
1947	Farrand, Robert E., Saugus	Tufts
1947	Feldman, Joseph D., Boston	Long Island College of Medicine
1946	Feresten, Morris, East Brewster	Tufts
1947	Fisher, H. Bernard, Dorchester	Tufts
1947	Fitzgerald, James A., Brookline	Harvard
1947	Fleming, William L., Newtonville	Vanderbilt University
1947	Fletcher, Kenneth S., Jr., Chicopee Falls	Boston University
1946	Ford, Richard, West Roxbury	Harvard
1947	Foster, George B., Jr., Cambridge	Jefferson Medical College
1946	Fox, Henry M., Wellesley Hills	Johns Hopkins University
1946	Frechette, Alfred L., Westwood	University of Vermont
1946	*French, Gordon N., Newton Centre	Tufts
1946	Freni, D. Richard, Swampscott	Tufts
1947	Fryer, Julius W., Danvers	University of Vienna
1947	Garland, Donald M., East Braintree	Tufts
1946	Gelfman, Raymond, Springfield	Tufts
1947	Gellis, Sydney S., Cambridge	Harvard
1947	Gesing, Emil J., Lawrence	Kansas City University of Physicians and Surgeons
1946	*Gevalt, Frederic C., Jr., Boston	Columbia University
1946	Gianturco, Nicholas D., East Boston	Middlesex
1946	Glicklich, Earl A., Brighton	University of Cincinnati
1947	Glickman, Abraham J., Dorchester	University of St. Vladismir
1946	Goldberg, M. Milton, Melrose	Tufts
1946	Golden, Isaac, Chelsea	Middlesex
1946	Goldfarb, Simon L., Northampton	University of Milan
1946	*Goldson, Robert J., Mattapan	Georgetown University
1947	Goodstone, Samuel B., Plymouth	Harvard
1946	Gorday, Walter J., Harvard	Tufts
1946	Gould, Maxwell E., Worcester	Middlesex
1947	Graham, John B., Newton Centre	Harvard
1946	Grant, W. Morton, Winchester	Harvard

1946	Groden, Harold M., Cambridge	Tufts
1947	Grossman, Myer J., Athol	Middlesex
1946	Grover, Morris L., Springfield	Tufts
1947	Guccione, I. Joseph, Roslindale	Middlesex
1947	Gucker, Thomas, Brookline	University of Pennsylvania
1946	Guild, Sherley A., Grafton	Tufts
1947	*Guterman, Burte, Worcester	Washington University
1947	Ham, Donald P., Greenfield	Boston University
1947	*Hanchett, Richard B., Auburndale	Tufts
1946	Harris, Oliver J., Boston	Tufts
1947	Haselhuhn, Donald H., Springfield	Tufts
1947	Hasenbush, Lester L., Dorchester	Johns Hopkins University
1946	Hastings, Nelson, Brookline	Harvard
1946	Hawn, Clinton V., Cambridge	Harvard
1946	Hayden, Charles G., Brookline	University of Minnesota
1946	Hecht, Paul L., Braintree	University of Freiburg
1947	Heifetz, Frank M., Lowell	Tufts
1946	Helman, Milton E., Chelsea	Boston University
1947	Hepburn, James P., Milton	Tufts
1946	Hermanson, Robert H., Brookline	Tufts
1946	Hill, Allen M., Beverly	University of Rochester
1946	Hinton, Elmer E., Boston	University of Kansas
1946	Hirsch, Lawrence S., Framingham	Middlesex
1946	Hoffman, John L., Cambridge	University of Buffalo
1947	Hormell, Robert S., Melrose	Harvard
1946	*Horwitz, William H., Belmont	Johns Hopkins University
1947	Hunter, John J., Cambridge	Tufts
1947	*Hunter, Richard E., Worcester	Boston University
1946	*Irving, Eliot S., Newton Highlands	Long Island College of Medicine
1946	*Isaacson, Philip A., Fitchburg	Dalhousie University
1947	Itkin, Irving H., Worcester	Indiana University
1946	Jaslow, Irwin A., New Bedford	Tufts
1946	Jewett, Everett P., Jr., Worcester	Tufts
1946	Jolliffe, Leslie S., Andover	Queen's University
1947	Jonas, Norman W., Pocasset	Chicago Medical School
1947	Judson, Harry E., Pittsfield	Syracuse University
1947	Kagan, Samuel, Brookline	Midwest Medical College
1946	Kaldeck, Robert, Lowell	University of Vienna
1946	Kaldeck, Rudolph, Dorchester	University of Vienna
1946	*Kaplan, Melvin S., Wellfleet	Albany Medical College
1947	Karpati, Oscar, Brookline	Royal Hungarian University
1946	Karpawich, Peter P., Worcester	Hahnemann Medical College
1946	Kaufmann, William, Holyoke	University of Geneva
1946	Kelley, Thomas F., Brookline	Boston University
1946	*Kelly, Arthur N., Waltham	Tufts
1946	Kennan, Fred J., Boston	Tufts
1946	Kennedy, Arthur P., Lowell	Tufts
1946	Kickham, Edward F., Brookline	Tufts
1946	Kilgore, Philip E., Lynn	Tufts
1946	Killam, Arthur R., Winchester	Boston University
1946	King, Myron N., Winthrop	University of Vermont
1946	Klestadt, Walter D., Fall River	University of Munich
1947	Knapp, Allen H., Quincy	Yale
1947	Krasner, George D., Quincy	University of Basel
1947	Lahey, Philip J., Worcester	Tufts
1946	Lamb, Marshall A., Winchendon	Tufts
1946	Larchez, Albert R., South Hamilton	Boston University
1946	Larcom, Rodney C., Jr., Dedham	Harvard
1946	Lareau, Henry R., Spencer	Boston University
1946	LaVigne, Richard J., Worcester	Tufts
1946	Lavoie, Robert J., Worcester	Hahnemann Medical College
1947	Leani, Aldo, Springfield	University of Vermont
1947	Leavitt, Joseph S., Malden	University of Lausanne
1946	Lenson, Norman, New York	Tufts
1947	Lent, Sylvester M., Hinsdale	Middlesex
1947	Leonard, Field C., Brookline	Harvard
1946	Lepreau, Frank J., Jr., New Bedford	Harvard
1946	Levenson, Herbert M., Brighton	Boston University
1946	Levine, Albert, Mattapan	Middlesex
1946	*Levine, Reevan I., Dorchester	Tufts
1946	Levinson, Leon, Newton Highlands	Tufts
1946	Levreault, Gerald V., Newton Centre	Tufts
1946	Lewenstein, Howard J., Brookline	Tufts
1946	*Lewis, Herbert D., West Newton	Harvard
1947	Lightman, Mashe U.L., Lowell	Middlesex
1947	Lipman, Daniel G., Lynn	Middlesex
1947	Livingstone, Robert G., Boston	Harvard
1947	Lord, William J., Great Barrington	Albany Medical College
1946	Loth, Eric C., Jamaica Plain	Middlesex
1946	Luongo, Angelo J., Revere	Middlesex
1946	Lydon, Roy T., Norwood	Tufts

1947	Lyle, Walter I., Wareham	Middlesex
1946	Lynch, Alice D., Dorchester	Tufts
1947	Lyons, Melvin K., Dorchester	Boston University
1947	Magwood, Robert W., Lynn	Tufts
1946	Maislen, Arthur A., Pittsfield	University of Vermont
1946	Maloof, Emil G., West Roxbury	Middlesex
1946	Manconi, Salvatore A., Revere	Boston University
1947	Mandeville, Ernest A., Holyoke	Tufts
1947	Manelis, Samuel R., New Bedford	Boston University
1947	Mann, Harold E., Dorchester	University of Pennsylvania
1947	Masterson, James H., Worcester	Tufts
1947	Matloff, Jacob, Brighton	Boston University
1947	Matz, Myron H., Dorchester	University of Minnesota
1947	McArdle, John J., Lawrence	New York University
1946	McArthur, Janet W., Minnesota	Northwestern University
1946	McCombs, Robert P., Brookline	University of Pennsylvania
1946	*McDonough, Walter J., Brighton	St. Louis University
1946	McIver, John M., Hingham	Boston University
1946	McKeigue, John E., Boston	Jefferson Medical College
1947	McKenna, Harold J., Fall River	Creighton University
1946	McKinnis, Peter P., Lawrence	Middlesex
1946	McLaughlin, Laurence S., Woburn	Tufts
1947	Merola, Joseph F., Waltham	Middlesex
1947	Merrick, Edward M., Milton	Middlesex
1947	Metcalf, Roger G., Southbridge	University of Rochester
1946	Mezer, Robert R., Newton Centre	Tufts
1947	Michell, Leon G., Lynn	Johns Hopkins University
1947	Middlebrook, Gardner, New York	Harvard
1946	Millen, Morris H., North Weymouth	Middlesex
1946	Miller, Harold, Chelsea	Midwest Medical College
1947	Miller, Harold I., Dorchester	Boston University
1947	Miller, Julius Y., Allston	Tufts
1946	Mitchell, Ralph J., Vineyard Haven	Vanderbilt University
1946	Monaghan, Leo B., Concord	Tufts
1946	Moore, Kenneth T., Hanover	Hahnemann Medical College
1947	Moriarty, Daniel J., Worcester	University of Vermont
1947	Morris, Lloyd E., Longmeadow	Ohio State University
1946	Morrison, Benjamin G., Northampton	Long Island College of Medicine
1946	Morrison, Herbert S., Brookline	McGill University
1947	Morrison, John L., Waltham	University of Pennsylvania
1946	Morse, Lawrence S., Brookline	Tufts
1946	Mosher, Henry A., Belmont	Northwestern University
1947	Mosig, William, Springfield	New York University
1947	Mucci, Alfred C., Somerville	Middlesex
1947	Mulvey, William A., Gloucester	Georgetown
1946	Myerson, Paul G., Boston	Harvard
1946	Naumann, Hans N., Taunton	Berlin University
1946	*Nereo, Oswaldo A., Quincy	Boston University
1947	Nochimow, Eliezer, New Bedford	Midwest Medical College
1946	O'Brien, Joseph A., Dorchester	Tufts
1947	O'Brien, Paul A., Winthrop	Tufts
1946	O'Brien, Paul I., Dorchester	Tufts
1946	O'Connell, William T., Roslindale	Tufts
1947	O'Day, John J., Norwood	Tufts
1946	O'Hara, John L., Newton	Tufts
1946	*Olive, George M., Jr., Belmont	Tufts
1947	Orchard, Norris G., South Harwich	Johns Hopkins University
1946	Osborne, Melvin P., Newton Highlands	Harvard
1947	O'Shea, James A., Lawrence	Long Island College of Medicine
1946	Owen, Charles K., Pittsfield	Tufts
1947	Pandolfino, Joseph E., Boston	Tufts
1946	Park, Irving H., Brookline	Middlesex
1946	Parnes, Jacob, Leominster	University of Prague
1946	Paull, Thomas, Boston	Harvard
1946	*Pavlo, Irving L., Cambridge	Harvard
1946	*Pennell, Walter J., Winchester	Harvard
1946	*Peretsman, Jacob, Somerville	Tufts
1946	*Perrone, S. Joseph, Worcester	Tufts
1947	*Pier, Arthur S., Jr., Milton	Harvard
1946	Pipi, John, East Boston	Middlesex
1947	Pirone, Francis A., Lynn	Tufts
1946	Powers, Hazel C. K., Boston	Middlesex
1946	Powers, Joseph W., Roxbury	Kansas City University of Physicians and Surgeons
1947	Powers, William J., Jr., Sharon	Tufts
1946	Prescott, Blake D., Connecticut	Middlesex
1947	Price, William B., Roxbury	Meharry Medical College
1946	Putnam, Henry M., Westwood	Harvard
1946	Putnam, Robert M., Plymouth	McGill University
1947	Quinn, Edward M., Jr., Lowell	Tufts
1946	Radcliffe, James, Jr., New Bedford	Yale
1946	Radovsky, Everett S., Fall River	Middlesex

1946	Record, Eugene E. Boston	McGill University
1946	Reder, Benjamin,, Fall River	Boston University
1946	*Regan, John E., Roslindale	Tufts
1946	Riccardi, Louis S., Marlboro	Middlesex
1946	Rizzo, Nicholas D., Brookline	Boston University
1946	Robinson, George E., Fall River	College of Physicians and Surgeons, Boston
1947	Robinson, Henry S., Somerville	Kansas City University of Physicians and Surgeons
1946	Roodin, Harry, Roxbury	College of Physicians and Surgeons, Boston
1947	Rosen, Abraham E., Worcester	Tufts
1946	Rosmatin, Ernest, Boston	University of Vienna
1947	Rossman, Benny, Brookline	University of Marburg
1947	*Ruben, Maurice, Dorchester	Boston University
1946	Rupp, John J., Boston	Washington University
1947	Russell, Donald H., Peabody	New York University
1947	Ruzicka, Edwin R., Chestnut Hill	University of Maryland
1946	Ryan, John J., Maryland	Tufts
1947	Saba, Edward, Lowell	Tufts
1946	*Sabino, Anthony W., Newtonville	Tufts
1947	Sanborn, Earl B., New York	Northwestern University
1947	Sarkisian, Sarkis A., Bridgewater	Boston University
1947	Scanlon, Joseph C., Worcester	Boston University
1946	Schwartz, Isaac H., New Bedford	University of Basel
1946	Scricco, Michael W., Worcester	Albany Medical College
1946	Sewall, Edgar F., Jr., Somerville	Tufts
1946	*Shalek, Seymour R., New York	Boston University
1946	Shand, Nathaniel K., Fall River	Middlesex
1947	Shane, Theodore, West Newton	Middlesex
1946	Shapiro, Eli, Roxbury	Middlesex
1947	Sharples, Arthur B., Hyannis	University of Vermont
1946	Sharry, Charles F., Cambridge	Harvard
1947	Shea, Daniel F., Quincy	Georgetown University
1946	Sheehan, John C., Marshfield	Tufts
1947	Sheehan, John F., Worcester	Tufts
1947	Siegel, Robert, New Bedford	Tufts
1946	Sikorsky, Lucy N., Grafton	Boston University
1947	Simoneau, Arthur G., Marlboro	Boston University
1946	Snyder, Eugene, Chicopee Falls	University of Prague
1946	Sommers, Sheldon C., Newton Centre	Harvard
1947	*Sozanski, Julius C., Peabody	Loyola Medical School
1946	Sprague, Marion L., Worcester	College of Medical Evangelists
1947	Stanley, Edith D., Brookline	Temple University
1947	Stanton, Joseph R., Newton	Yale
1947	*Stanton, Richard H., Boston	Harvard
1946	Starr, Steven J., Worcester	Georgetown
1946	Steinberg, Irving H., Northampton	Medical College, State of South Carolina
1946	Steinman, Solomon E., Brighton	Boston University
1947	Stevenson, Stuart S., Boston	Yale
1946	Stone, Chauncey M., Jr., Arlington	Boston University
1946	Strauss, Elliott G., Northampton	Tufts
1947	Strauss, Louis, Dorchester	Middlesex
1947	Sullivan, Arthur P., Quincy	Tufts
1946	Sullivan, Frederick J., Jr., Fall River	Jefferson Medical College
1947	Sunshine, Samuel, Milton	Middlesex
1946	Swank, Roy L., Newton Highlands	Northwestern University
1946	Swartz, Morris, Dorchester	Boston University
1947	Sweetser, Elliott H., Malden	Boston University
1947	Thompson, Charles A., Newton Highlands	Tufts
1946	*Thompson, Robert C., Swampscott	Syracuse University
1946	Tierney, Thomas M., Brighton	Georgetown
1946	Toppenberg, David R., Melrose	College of Medical Evangelists
1946	*Traina, Salvatore R., East Boston	Tufts
1946	Tribby, William W., Brookline	Harvard
1946	Trodella, George P., Somerville	Tufts
1946	True, Ansel B., East Northfield	Yale
1947	Tucker, Walter I., Boston	Harvard
1946	Tullis, James L., Newton	Duke University
1946	*Vecchione, Felix S., East Boston	Boston University
1946	Viterbi, Achille, Boston	University of Turin
1946	Waite, Harold V. M., Easthampton	Middlesex
1946	Warren, Kenneth W., Newton Highlands	Temple University
1946	Webster, Richard C., Jr., Burlington	Harvard
1947	Weinberger, Jerome L., Boston	New York University
1947	Weisman, William S., Mattapan	Middlesex
1946	Welch, C. Stuart, Brookline	Tufts
1946	Welch, William J., Taunton	Tufts
1947	Wharton, Russell S., Northampton	University of Arkansas
1946	*White, Malvin F., Cambridge	Yale
1947	Whitman, Elmer L., New Bedford	Tufts
1947	Whitney, Karl R., South Sudbury	Tufts
1946	Wilhelm, Norbert A., Newton Centre	St. Louis University
1946	Williams, Nathan, Allston	Boston University

1947	Willis, Albert W., Lowell	Tufts
1947	Wilson, Franklin L., Stockbridge	Middlesex
1946	Wise, Herman R., Roxbury	Middlesex
1947	Wojciechowski, Anthony A., Webster	Tufts
1946	Wolfe, Louis M., Chelsea	New York University
1947	Woll, Ephraim, Boston	Creighton University
1946	Woods, Francis M., Newton Highlands	Yale University
1946	Woodworth, Clyde R., Peabody	Boston University
1946	Worcester, John, Waban	Tufts
1947	Worthington, Richard V., Framingham	Yale University
1947	Wright, Walter E., Orleans	Cornell University
1947	Wyman, Stanley M., Cambridge	Harvard
1946	York, Richard F., Waban	Middlesex
1947	Zambella, Joseph, Winthrop	Kansas City University of Physicians and Surgeons
1947	Zovickian, Anthony, Watertown	Yale

*The candidate, after a personal interview, was approved by the Committee on Membership and permitted to take an examination before a board of censors.

Deaths Reported from May 22, 1946 to May 20, 1947

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1894	Adams, Charles Sumner	Wollaston	June 13, 1946	76
1916	†Alley, Ernest J.	Billerica	May 23, 1946	72
1911	Almy, Thomas	Fall River	September 9, 1946	63
1894	†Bailey, Marshall H.	Norwell	September 18, 1946	87
1908	Barker, Williston W.	Dorchester	November 26, 1946	64
1925	Bergeron, George G.	Ludlow	September 26, 1946	55
1883	†Bigelow, Enos H.	Framingham	March 13, 1947	91
1903	Boyle, John F.	Lowell	April 25, 1947	76
1942	Burger, Franklyn D.	Wellesley Hills	November 25, 1946	40
1924	Caldicott, Francis S.	Lowell	January 17, 1947	57
1914	Caswell, Bertram H.	West Somerville	April 14, 1947	72
1915	Chandler, Harold B.	West Newton	July 14, 1946	61
1907	Chase, Harrison A.	Brockton	July 23, 1946	69
1886	†Cheever, Clarence A.	Scituate	November 31, 1946	88
1910	Clarke, Joshua W.	Attleboro	March 27, 1947	76
1919	Clute, Howard M.	Boston	September 19, 1946	56
1903	Collins, Richard	Waltham	July 28, 1946	72
1902	Conro, Arthur C.	Boston	August 18, 1946	76
1909 1943	Crosbie, Arthur H.	Boston	August 23, 1946	69
1903	Crosby, Leander M.	New York	August 18, 1946	70
1926	Davis, Max	Boston	April 30, 1947	47
1905	Day, Hilbert F.	Cambridge	May 17, 1947	68
1900	†Dennett, Alonzo G.	Lowell	December 11, 1946	91
1932	Djerf, Frederick J.	Fitchburg	July 15, 1946	45
1892 1920	†Dole, Mary P.	Shelburne	March, 1947	84
1905	Dwyer, William J.	Boston	September 30, 1946	63
1905	Ellis, Edward K.	Needham	June 7, 1946	67
1915	Ellison, Daniel K.	Lowell	February 15, 1947	60
1934	Ellms, Evelyn B.	Waban	July 14, 1946	40
1921	Emery, Edward S.	Brookline	March 16, 1947	53
1943	Ewing, Edward H.	Stoughton	March 8, 1947	74
1943	Gigger, Augustus G.	Falmouth	November 27, 1946	68
1891	†Godfrey, Joseph W.	Lynn	November 27, 1946	79
1919	†Grant, Justin F.	Boston	1947	70
1888	†Hare, Charles H.	Boston	January 21, 1947	84
1935	Harpin, Raymond A.	Lynn	August 10, 1946	41
1926	Hayes, Arthur W.	Montague	March 11, 1947	55
1926	†Hayes, Frederick L.	Brookline	July 7, 1946	77
1901	Hills, Charles E.	South Natick	February, 1947	78
1902	Holbrook, Bradbury	Waltham	August 24, 1946	74
1944	Holt, William L.	Amherst	October 18, 1946	68
1919	Howard, Perez B.	Newtonville	1947	71
1908	†Howland, George L.	Jamaica Plain	August 7, 1946	76
1936	Huber, Edward G.	Waban	July 23, 1946	64
1878	Hunt, William O.	North Falmouth	April 19, 1947	93
1895	Hutchinson, Charles M.	Cambridge	November 26, 1946	76
1921	Jennings, John G.	Weston	February 27, 1947	58
1914	†Johnson, Herbert L.	Hadley	December 22, 1946	87
1917	Kane, William V.	Lynn	December 11, 1946	54
1939	Kees, Philip A.	—	1946	39
1893	Kelly, William P.	Pittsfield	April 15, 1947	76
1911	Kerrigan, Joseph H.	Miami, Florida	January 3, 1947	61
1913	Kilburn, Ira N.	Springfield	February 3, 1947	62
1930	Kingsley, Frederick	Chestnut Hill	February 1, 1947	—
1900	Kinney, William D.	Osterville	July 12, 1946	73
1925	Landesman, Henry M.	Boston	May 22, 1946	62

1910	Lee, Harry J.	Boston	August 7, 1946	65
1912	Marsden, George	New Bedford	May 20, 1946	68
1932	McCarthy, Charles K.	Webster, Iowa	1945	50
1924	McNamara, John J.	Lowell	November 13, 1946	51
1926	Meserve, Faith L.	Weston	July 19, 1946	52
1911	Morgan, Charles R.	Medford	November 28, 1946	68
1916	Moulton, Allen T.	Boston	November 22, 1946	56
1932	Musso, George H.	Lynn	March 1, 1947	73
1906	†Noble, Mary G.	Roxbury	February 16, 1947	75
1922	O'Brien, Thomas F.	Worcester	September 16, 1946	51
1919–1934	O'Dea, Patrick J.	Fitchburg	September 25, 1946	59
1925	O'Halloran, William T.	Newton	March 21, 1947	46
1919	O'Hara, Francis J.	North Adams	May 2, 1947	69
1910	†Overlander, John E.	Springfield	May 26, 1946	65
1896	†Painter, Charles F.	Brookline	January 6, 1947	77
1892	†Patch, William T.	——	February 7, 1947	85
1921	Perkins, George E.	Fitchburg	April 1, 1947	69
1892	†Phelps, John S.	Lynn	December 3, 1946	80
1907	Phillips, Wilson F.	Dorchester	January 29, 1947	74
1884	†Preble, Wallace	Cambridge	February 22, 1947	89
1910	Pulsifer, Walter H.	Whitman	September 26, 1946	62
1909	†Quest, James F.	Boston	May 27, 1946	68
1907	Reid, Isadore E.	Jamaica Plain	August 12, 1946	72
1896	Richardson, Mark W.	——	February 12, 1947	79
1916–1942	Roney, Hugh B.	Pittsfield	January 8, 1947	61
1931	Rose, Wilfred A.	San Francisco	September 24, 1946	41
1901	Ryder, George H.	Quincy	March 12, 1947	72
1893	Sargent, Ara N.	Salem	August 26, 1946	78
1917	Sedgley, Frank R.	California	February 12, 1947	67
1929	Shefferd, Jeannette M.	——	October 25, 1946	—
1926	Simmonds, Frederick J.	Jamaica Plain	December 6, 1946	68
1930	Smith, Roswell H.	Edgartown	February 12, 1947	52
1892	†Stacey, Charles F.	Maine	July 18, 1946	80
1909	Stone, George H.	Worcester	January 4, 1947	65
1914	Stone, Henry E.	Dorchester	March 31, 1947	58
1942	Sullivan, Arthur J.	Fall River	August 6, 1946	56
1942	Sulzbach, Wolfgang M.	Boston	1946	37
1905–1925	†Sylvester, Albie W.	Pittsfield	June 20, 1946	83
1944	Taylor, Lois E.	Belchertown	March 21, 1947	37
1928	Terry, Theodore L.	Stow	September 28, 1946	47
1908	Tighe, Michael A.	Lowell	April 8, 1947	65
1899	†Tilden, Irving N.	Mattapoisett	January 29, 1947	74
1906	Tinkham, Oliver G.	Newton	September 10, 1946	67
1901	Tozier, Charles H.	Winchester	January 1, 1947	72
1910–1946	Tynan, Joseph P.	——	1947	63

†Retired fellow.

Total number of deaths of active fellows	71
Total number of deaths of retired fellows	22
Grand total	93

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 33351

PRESENTATION OF CASE

A forty-six-year-old man entered the hospital because of upper abdominal pain.

There was a life-long history of "stomach ache" three or four times a year. Six years before admission the patient suddenly and painlessly vomited over a cupful of red blood. He was put to bed and treated with Amphogel and belladonna and placed on a bland diet. From that time he began to have gnawing epigastric pain every two or three weeks, lasting one or two days at a time. The pain responded readily to milk, to sodium bicarbonate or simply to a stricter adherence to the diet. Three and a half years before admission the pain became worse and failed to respond to the medical regime. X-ray examination of the stomach was said to have shown an ulcer that was "partially blocking the outlet." An operation, probably a gastroenterostomy, was performed with complete relief for a year and a half. Ten months before admission, after a series of family misfortunes, the attacks returned. At that time they were even severer, occurring several times a week and radiating from the epigastrium to the back. Sodium bicarbonate afforded immediate relief. Six months before entry the patient had an attack of "intestinal grippe," with a pain and cramps low in the abdomen, and diarrhea without blood or mucus lasting for a week. The pain in the upper abdomen was subsequently constant, soda affording relief for only twenty minutes at a time. The patient went on a self-imposed milk and egg diet and lost 16 pounds without much benefit. He could not recall specific instances of food aggravating the pain, but had an impression that eating normal meals was deleterious. Throughout that period he never felt nauseated and had noted no change in bowel habits.

Physical examination revealed a thin patient, who complained of gnawing epigastric and back pain until relieved by milk. The heart and lungs were normal. A vertical scar occupied the midline in the epigastrium. The epigastric region was slightly tender.

The temperature was 99.5°F., the pulse 88, and the respirations 16. The blood pressure was 120 systolic, 74 diastolic.

Examination of the blood disclosed a red-cell count of 4,250,000 and a white-cell count of 14,500, with 80 per cent neutrophils. The urine was normal. The stools gave a ++ reaction for blood.

On fluoroscopic examination, the stomach was seen to be filled with a large amount of residual food. Along the lesser curve there was a large area of rigidity, with an ulcer crater 2 cm. in depth and 3 cm. in diameter within which there was a filling defect (Fig. 1). The lesser curvature, from a distance of about 2 cm. above the ulcer down to the pylorus, was rigid and shortened. Insufficient barium passed into the duodenum to fill out the duodenal bulb to a normal contour. No barium left the stomach by way of an anastomosis.

A fasting gastric aspiration failed to produce free acid, which was present after the administration of histamine, however.

The patient was kept virtually free of pain by hourly feedings. The temperature and white-cell count fell to normal. The morning gastric residual fluid was reduced from 300 to 75 cc. during the first week. On the seventeenth day gastroscopy revealed a constantly deformed and rigid-appearing angulus whose outline was irregular and slightly nodular. No peristalsis was visible. The ulceration seen at fluoroscopy was out of the range of the gastroscope.

Three days later an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD SCHATZKI: I shall reverse the customary procedure and start with the x-ray films. That is my usual approach to a case, although I have the history in the back of my mind while discussing the films. The record gives an adequate description of what is seen on the films. There is a crater. One thing that I cannot see but that the fluoroscopist apparently saw is the pylorus, unless it is this area, which would mean that the crater extended all the way to the pylorus. The lesion is characterized by a large crater that differs from the usual crater in that it contains a filling defect. The rigidity described in the record is not visible on the film. The shortening of the lesser as compared to the greater curvature can be seen. Another point, not mentioned in the record, is that the gastric folds converge to this crater. That does not help in differential diagnosis, however, because we have seen too many cancers with converging folds. Some of the folds reach the crater, but others do not quite reach it. That may be of significance.

The differential diagnosis, of course, is between benign and malignant ulcer. I might say that if the roentgenologist can be of any help in the differential diagnosis it is in the type of case in which he can see a tumor that has become ulcerated. The ulceration may be the leading roentgenologic sign, but by careful examination the tumor that has ulcerated and surrounded the ulcer may be demon-

strated. In such a case the roentgenologist is helpful because he can make a flat-footed diagnosis of cancer. There are other cases in which the lesion has the characteristic appearance of what we learned in school to be peptic ulcer of the stomach. In a case like the one under discussion the roentgenologist can say that the ulcer is macroscopically benign but cannot say that the lesion is not malig-

FIGURE 1.

nant histologically. I shall not go into statistics regarding how many of these lesions turn out to be cancer on microscopical examination. A third type of lesion — these are the difficult ones — occurs in cases in which the ulceration is present in an infiltrating cancer. The cancer does not form a real tumor mass but merely a small, nodular irregularity around the crater. In the case under discussion I cannot see anything that I should call a gross polypoid tumor that may have ulcerated. Offhand, my first impression was to call this a grossly benign ulcer from the x-ray point of view. There are a few disturbing facts about such a statement, one of which is that the folds do not quite reach the crater. This may be seen in the ulcerative infiltrating type of cancer. Another reason is this peculiar defect in the crater. After having looked at the defect for some time I decided that I still did not know what it was. I thought of a polypoid tumor protruding into the lumen, which I cannot remember ever having seen and which I do not believe this defect represented. I am certainly not able to say whether an anastomosis was present, since none is visible. One reason for its not being visible may be that the anastomosis was closed off; another possibility is that the operation three and a half years before entry was not a gastroenterostomy done on the lower pole of the stomach but a pyloroplasty. In that event the appearance of the crater might have had something to do with the operative procedure. There was no history of bleeding in this case to warrant the diagnosis of blood clot. Food in the ulcer may give such an appearance, although the defect seems too constant to be explained on the basis of food. It may be worth while to say a few words about the clinical record.

I believe that the history alone in cases of this sort does not indicate the differential diagnosis between benign and malignant ulcers, but it increases the percentage of probability one way or the other. When I started reading the history I had no doubt that the patient had had a benign ulcer in the past, at either the pylorus or the duodenum. The course sounds typical of a classic case of ulcer. Then, in the last year, something happened. The history becomes confusing. If one did not have the first part of the record one might think that this was a case of cancer. The pain was relieved by milk. I do not know how much attention is paid these days to gastric acidity. It can certainly be said that patients with cancer may have a great deal of gastric acid and that patients with ulcer may have no acid at all, although in a large group of cases patients with ulcer have more acid than those with cancer. In a case like the one under discussion the presence or absence of acidity does not make the diagnosis; it merely changes the percentage. This man had some acid after the administration of histamine.

How does the size of the ulcer enter the picture? It does not, as a matter of fact. If there is a large crater, which appears grossly benign, the chances are that histologically it will be benign. The cases in which there is difficulty in the differential diagnosis are those of small ulcers, 1 or 2 cm. in diameter. I shall not discuss the significance of the location of the ulcer in detail and shall say only that a great many ulcerations in the prepyloric areas are cancers and that a great many are benign.

Why, in a given case, does one bother to talk about the differential diagnosis — why not remove the stomach if the treatment of gastric ulcer is definitely surgical? It is true that in practically none of these cases can histologic malignancy be ruled out, but given a lesion that looks grossly benign in a patient who is a poor operative risk, one should be more conservative in treatment. For this reason it is justifiable and essential to make the preoperative diagnosis as exact as possible. In the case in question the chances are that the lesion was grossly benign, but it may have been an ulcerated infiltrated cancer. This is not the type of ulcer that I should entrust to medical treatment.

Dr. Benjamin Castleman: The report of Dr. Schulz was as follows: "The appearance is that of a large gastric ulcer that has more of the criteria of a grossly benign ulcer than of a malignant one. Cancer cannot be ruled out. The area is fixed."

Dr. Welch, have you any opinion?

Dr. Claude E. Welch: I should think that it was cancer. If you ask me why, I cannot tell you.

Clinical Diagnosis

Gastric ulcer.

Gastric carcinoma?

Dr. Schatzki's Diagnosis

Grossly benign ulcer.

Anatomical Diagnosis

Multiple benign peptic ulcers of stomach.

Pathological Discussion

Dr. C. J. Mixter, Jr.: This man had had a previous posterior gastroenterostomy, and at operation there was a large mass in the region of the prepyloric area that grossly appeared to be malignant. There was also evidence of an old duodenal ulcer. The duodenum was closely drawn together, so that the acute lesion was really a prepyloric ulcer. Since the lesion appeared to invade the pancreas beneath it, we thought that it was necessary to treat it as a malignant lesion and took out part of the pancreas beneath it.

Dr. Castleman: The specimen that we received showed a firm, brawny mass in the lower third of the stomach. The fat around it seemed to be porky. When the stomach was opened the center of the mass showed an ulceration about 4 cm. in diameter. We were unable to find anything to correspond to the filling defect. One of the surgeons who saw it said that the base was flat and granular and that there had been no projections from it at the time of operation. There may have been something there when the x-ray film was taken. Just distal to the ulcer was an opening of a gastroenterostomy on the posterior wall, and there was also another ulcerating, rather shallow lesion on the lesser curvature. On microscopical examination the large ulcer was benign, with a base that was made up primarily of the serosal fat. There was practically no stomach left. The other lesion high up on the stomach was also a benign ulcer.

Dr. Schatzki: Do you remember how large the upper ulcer was?

Dr. Castleman: Less than 1 cm. in length and somewhat shallower in depth — I should say, from the microscopical sections, not more than 0.5 cm.

CASE 33352

Presentation of Case

First admission. A sixty-one-year-old American-born Irish plumber was admitted to the hospital because of abdominal swelling.

About two months before admission the patient had noticed the onset of a dull abdominal discomfort centering in the epigastric and periumbilical regions. It was a steady, dull pain occurring during the morning or early afternoon and tending to persist throughout the day. It was sometimes precipitated or aggravated by food or activity. There was no particular way to relieve the pain. The patient had also noticed bloating accompanying meals, and for six weeks prior to admission the abdomen had

become increasingly swollen, causing some dyspnea. He began to be constipated a month before admission, the previous bowel habits having been regular. Laxatives had produced daily bowel movements in that period, and the urine had been dark. During the week before admission he noted that the stools were somewhat lighter. Also for several weeks prior to admission he had noticed an increase in his chronic "cigarette cough," with the production of white phlegm, occasionally flecked with blood. He denied vomiting, hematemesis or tarry stools, although at an interview in the Out Patient Department two weeks prior to admission he had admitted vomiting blood some five weeks previously and passing a dark stool. He had not been jaundiced and had had no itching. He had continued to work until a month before admission.

Ten years previously an operation had been performed for a perforated peptic ulcer, without preceding or subsequent gastrointestinal symptoms until the present illness. After the operation the patient had followed a bland diet for five months, and he was then told that he was well. For at least ten years he had been drinking a pint of whisky and six or seven glasses of beer daily. During that time the diet had been fair, with three meals a day, red meat once daily, at least one vegetable and frequently fruit, until about six months before admission, when his appetite had become rather poor and he had begun to skip meals. He had had occasional epistaxis for many years, most recently on two or three occasions after blowing the nose. Also, the ankles had been swollen for about ten years, in association with the development of varicose veins. He denied any history of rheumatic fever or swollen joints.

Physical examination disclosed a well developed, poorly nourished, ruddy-appearing man in no acute distress, with many telangiectases and spiders over the shoulders, anterior upper portion of the chest and the arms. There was questionable icterus of the scleras, and slight injection of the nasal mucosa. The tongue had smooth, red edges. The heart appeared enlarged to the left and right. The apical impulse was 10 cm. to the left of the midsternal line in the fifth interspace. The apical first sound was loud and booming. The pulmonic second sound was louder than the aortic. There was a Grade II systolic, roaring murmur heard best in the third and fourth left interspaces and at the apex. The abdomen was greatly distended, with shifting dullness and a fluid wave. The liver was not enlarged to percussion, and neither the liver edge nor the spleen could be felt. External hemorrhoids were visible on rectal examination. There was a slight tremor of the hands, and Dupuytren's contractures of both hands. There was marked edema up to the mid-thigh, and moderate sacral edema. There was brown pigmentation over the lower legs bilaterally.

Examination of the blood revealed a red-cell count of 3,030,000, with 10 gm. of hemoglobin, and a white-cell count of 3300, with 69 per cent neutrophils. The urine gave a ++ test for albumin and a ++ test for bile, with rare hyaline casts in the sediment. The stools were guaiac negative. The prothrombin time was 28 seconds (control, 16 seconds). The total protein was 7.2 gm. per 100 cc., with an albumin of 3.0 and a globulin of 4.2 gm. (albumin-globulin ratio of 0.7); the nonprotein nitrogen was 22 mg., the cholesterol 170 mg. and the vitamin A 0.0 units per 100 cc., and the cephalin-flocculation test was +++ in twenty-four and ++++ in forty-eight hours. The van den Bergh reaction was 1.4 mg. per 100 cc. direct and 2.2 mg. indirect, and the alkaline phosphatase 2.7 units per 100 cc. The blood Hinton reaction was negative.

A gastrointestinal series revealed a small hiatus hernia at the lower end of the esophagus and varices in the esophagus above. The stomach appeared quite normal. Barium passed the pylorus without hesitation, filling the duodenal bulb, which was deformed in a manner characteristic of an old ulcer but within which no active crater could be demonstrated. Chest films showed prominence of the left ventricle and linear areas of atelectasis in both bases. There was elevation of both leaves of the diaphragm.

Mercupurin, with a high-protein, high-carbohydrate, low-fat diet, was administered, and two transfusions were given. Two and a half weeks after admission the total protein was 7.1 gm. per 100 cc., with an albumin of 2.3 gm. and a globulin of 2.8 gm. (albumin-globulin ratio of 0.9), the prothrombin time had dropped to 20 seconds (control, 15 seconds), and the cephalin-flocculation test remained unchanged, as did the van den Bergh reaction. A paracentesis three weeks after admission yielded 8350 cc. of cloudy, amber fluid with a specific gravity of 1.012. A bromsulfalein test at that time revealed 44 per cent retention of the dye. In spite of massive doses of vitamin K intravenously, the prothrombin time remained at 20 seconds against a 15-second control. It was believed that this contraindicated a liver biopsy.

A month after admission the temperature suddenly spiked to 103°F., following a shaking chill. The patient had a nosebleed that morning, and he stated that he had been getting blood out of the back of his throat for the past three days. He had a slight cough, no chest pain or dyspnea and no leg or abdominal pain. The throat was slightly injected. A chest film showed linear areas of increased density in the right lower chest. The right leaf of the diaphragm was elevated and somewhat irregular. By that night the temperature had receded, and the patient felt much better. A few days later a cephalin-flocculation test was ± in twenty-four hours and ++ in forty-eight hours, and the thymol turbidity was 5.5 units. The

icterus seemed definitely less. The hemoglobin was up to 13 gm. Ascites, however, seemed to reaccumulate rather rapidly. Five weeks after admission the patient vomited following nausea, although he had had almost nothing by mouth. He had one bowel movement of tarry, black, unformed material and seemed slightly confused, twitching frequently with gross clonic contractions of large muscle groups, but he showed no evidence of shock and the blood pressure and pulse remained steady. Subsequently, he passed two fairly large tarry stools. There was no change in the blood pressure or pulse and no hematemesis. Several transfusions were given, and the stools became brown but were still guaiac positive. There seemed to be little change in the course, and the bleeding had apparently stopped; consequently, the patient was discharged on a strict diet seven weeks after admission.

Second admission (six weeks later). During the interim the patient was followed in the Out Patient Department and apparently kept to his diet. He was given Mercupurin, and occasional paracenteses were done. After the last paracentesis he developed a dry cough and pain in the lower abdomen, especially when coughing. He had had a few small hemoptyses during the week prior to admission. He had felt badly, and his appetite had become poor. He suddenly developed a shaking chill, with a subsequent rise of temperature, and he was admitted to the hospital with a temperature of 104.5°F. and a blood pressure of 148 systolic, 84 diastolic. He was treated with penicillin and within three days showed great clinical improvement with subsidence of the fever. A paracentesis was performed on the fourth hospital day, yielding 12,700 cc. of deep-yellow, opalescent fluid with a specific gravity of 1.004. The patient was subsequently discharged.

Final admission (two weeks later). The patient was admitted in a slightly confused state. He stated that about a week before admission his appetite had begun to diminish and that he had started to vomit coffee-grounds material after meals. He was constipated for a week, and when he finally had a bowel movement on the day of admission the stool was copious and brown. He had observed no frank blood in the stools, which had not been clay colored. During that period he had complained of severe heartburn starting in the epigastrium and made worse by food. This pain radiated up to the anterior portion of the chest. Four days prior to admission the abdomen had become tight and swollen, and a physician had performed a paracentesis. The patient was given 4 gm. of ammonium chloride daily for a week.

Physical examination was essentially unchanged. Examination of the blood revealed a red-cell count of 3,000,000, with 10 gm. of hemoglobin, and a white-cell count of 8600, with 84 per cent neutrophils, 16 per cent lymphocytes and toxic granulation of the neutrophils. The urine was normal. The stools were guaiac negative. The carbon dioxide was 20.8 milliequiv. per liter, the prothrombin time 33 seconds (control, 20 seconds), the cephalin-flocculation test + in twenty-four and ++ in forty-eight hours, and the total protein 7.0 gm. per 100 cc., with 2.5 gm. of albumin and 4.5 gm. of globulin (an albumin-globulin ratio of 0.55). The nonprotein nitrogen was 52 mg. per 100 cc., the chloride 91 milliequiv. per liter, and the van den Bergh reaction 3.6 mg. per 100 cc. direct and 5.4 mg. indirect. The patient seemed to improve temporarily, but on the tenth hospital day he began vomiting large amounts of guaiac-positive, coffee-grounds material. There was some tenderness in the epigastrium, and finally bright-red blood began to appear in the vomitus. Repeated transfusions were given, but to no avail, and the patient died after semicoma and a period of vascular collapse on the fifteenth hospital day.

Differential Diagnosis

Dr. Vincent P. Dole: This patient presented a group of findings that are classic for cirrhosis of the liver. For this primary diagnosis we are given a history of alcoholism, symptoms of anorexia and epigastric discomfort, findings of varices, ascites and hemorrhoids indicating portal hypertension, spider angiomas, low-grade icterus and laboratory evidence of impaired liver function.

There is nothing in the history to suggest that biliary obstruction, neoplasm, infection or toxic agents damaged the liver. On the other hand, it is probable that the patient suffered from a deficient diet, since the stated intake of whisky and beer contributed over 3000 calories a day to a person reported as appearing poorly nourished. Had this caloric intake been supplemented by an adequate diet, he would surely have been obese. The observation of smooth, red tongue edges suggests deficiency of vitamin B factors.

The findings of cardiac enlargement and murmur require separate consideration. Part of the enlargement seems to have been apparent only and due to a transverse position of the heart from a high diaphragm. Allowing for this, there was possibly sufficient dilatation of the left ventricle to cause functional mitral regurgitation. Deficiency of thiamine may have been contributory. There is also a fair possibility that the patient had coronary-artery disease; an electrocardiogram might have been of interest. In any event, there was no engorgement of the neck or arm veins and presumably no tachycardia to suggest that cardiac failure was a factor in the ascites and leg edema.

The episodes of hemoptysis and fever could have been due to pulmonary infection, collapse or infarction. Tuberculosis is an occasional complication of cirrhosis, but the x-ray films are not suggestive of that diagnosis. Sputum examinations would

nevertheless have been desirable. The best explanation appears to me that of multiple pulmonary infarctions, to which the patient was disposed by thrombosis of congested deep leg veins.

Finally, one is struck by the rather abrupt appearance of severe portal hypertension with intractable ascites and death apparently from a ruptured esophageal varix within about five days. Although such a rapid progression after onset of ascites occurs in uncomplicated cirrhosis, one is nevertheless tempted to speculate on a complicating portal-vein thrombosis. The symptoms of abdominal distress are consistent with such a diagnosis but occur quite often in cirrhosis alone.

I am unable either to establish or to exclude the diagnosis of portal-vein thrombosis and must leave a question to be asked of the pathologist.

Clinical Diagnoses

Cirrhosis of liver, alcoholic type.
Rupture of esophageal varices.

Dr. Dole's Diagnoses

Portal cirrhosis.
Portal-vein thrombosis?
Multiple pulmonary infarctions.

Anatomical Diagnoses

Cirrhosis of liver, alcoholic type.
Duodenal ulcer, chronic, active, with erosion into pancreaticoduodenal artery.
Hemorrhage, recent, into intestines, stomach and esophagus.
Esophageal varices?
Ulcerations of esophagus.

Pathological Discussion

Dr. Tracy B. Mallory: The primary diagnosis in this case proved, as Dr. Dole predicted, to be cirrhosis of the liver. The organ was atrophic, weighing only 1050 gm. Its surface was coarsely granular, the granules ranging from 1 to 3 mm. in size, and it was diffusely tough and fibrous.

We found several possible sources for the attacks of gastrointestinal hemorrhage. The gross evidence of varices at the time of post-mortem examination was not impressive, but sections of the esophagus showed collapsed mucosal veins of considerably more than normal diameter. The mucosa of the esophagus, however, revealed numerous shallow linear ulcerations ascending from the cardia of the stomach for a distance of 10 cm. The epithelial layer had been completely destroyed in many areas, exposing an extensive capillary bed, and these excoriations could have been the source of significant bleeding. Finally, the old ulcer in the duodenum had become reactivated, and there was fresh peptic ulceration. It is quite possible that hemorrhage occurred at various times from all three of these factors. At autopsy the entire gastrointestinal tract from stomach to rectum contained blood. The spleen was only slightly enlarged, weighing 300 gm. In patients with extensive hemorrhage just before death, however, the spleen often shrinks rapidly, and it is probable that it was considerably larger during life.

We found no thrombosis in the portal system. The lungs were somewhat collapsed and markedly edematous.

We believe in this laboratory that portal cirrhosis of the liver can be divided into two types. The first of these follows one or more nonfatal attacks of diffuse necrosis or so-called "atrophy." The second is seen ordinarily only in patients with a history of chronic alcoholism and coincident dietary insufficiency. The latter is very readily recognizable in its diffuse progressive stages by the combination of fatty vacuolization of the liver cells and a peculiar and characteristic form of hyaline degeneration. Both these changes, however, disappear rather quickly under the influence of a high-protein diet. In serial biopsies we have been able to show that the fat has decreased by 50 per cent within a month and has almost entirely gone within two months. Consequently, these pathognomonic signs cannot be expected in a patient who has been under medical treatment for a significant period. In such cases we must rely on other criteria for diagnosis. The best of these is the gross appearance of the organ. In cases that follow subacute atrophy the regeneration is irregular and coarsely nodular, the individual nodules frequently exceeding 1 cm. in diameter and sometimes reaching 2 cm. The intervening bands of fibrous tissue are correspondingly wide and coarse. In the alcoholic type of cirrhosis, in contrast, the regeneration is much more diffuse, and the nodules are uniformly small, ranging from 1 to 4 mm. in diameter. The liver of this patient was clearly of the latter type.

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HOUSE OF DELEGATES

The report of the Massachusetts representatives to the June meeting of the House of Delegates of the American Medical Association, which will be presented to the Council of the Society at its October meeting, contains several matters of general interest.

In the first place, it comments on the final report of the committee to consider public relations and the liaison between the American Medical Association and the National Physicians Committee. The committee completely justified co-operation with the National Physicians Committee and recommended a change in the public-relations consultant, Rich Associates. The report was unanimously approved, and the recommendation adopted.

Secondly, the delegates' report calls attention to the emphasis throughout the meeting on the importance of the general practitioner to the community and the need for helping him in every way possible. This was brought to a climax when it was voted to hold the December meetings of the House of Delegates in various cities, rather than in Chicago, and to follow each meeting with a two-day scientific exhibit arranged for the sole benefit of the general practitioner. Furthermore, the Council on Medical Education and Hospitals advised that, to promote a resolution previously adopted by the House of Delegates whereby hospitals were urged to establish general-practitioner services, one section of "Essentials of a Registered Hospital" be revised to read, "Staff sections, such as medicine, obstetrics, surgery, general practice and so forth, should be organized as may seem wise." In addition, the Council stated that it did not approve of the habit of some hospitals, particularly those approved for resident training, in limiting staff appointments to physicians who are certified by one of the specialty boards; any physician, it added, who has had the training and experience approximately equivalent to that required for certification should be capable of rendering satisfactory service.

Thirdly, the report places emphasis on an address given by the Secretary of War, Robert P. Patterson. Mr. Patterson outlined briefly a bill (H.R. 3174, S. 1143) designed to promote and enhance the professional standing of the Army Medical Corps, thus placing it on a level equal to that in the best civilian practice. This bill contains the following provisions: an increase in base pay for all medical officers with less than thirty years' service; a 25 per cent increase in base and longevity pay for all officers who are certified as specialists; the appointment of outstanding medical specialists from civilian life to grades above those now permitted; the appointment of four physicians, distinguished respectively in the fields of medicine, surgery, neuropsychiatry and preventive medicine, as professors, with the rank of brigadier or major general, to supervise professional standards, educational programs and the assignment of qualified officers; and the utilization, if necessary, of civilian physi-

cians on a temporary part-time or full-time basis, without regard to Civil Service requirements. In closing, Mr. Patterson stated that the War Department would do everything within its power to establish, maintain and foster a high grade of professional attainment within the Medical Corps and requested support of the legislation and the program in general.

At the final session of the House of Delegates, Dr. R. L. Sensenich, of South Bend, Indiana, was made president-elect, and Dr. E. J. McCormick, of Toledo, Ohio, was appointed a member of the Board of Trustees. The annual meetings of the Association in 1948 and 1949 will be held in Chicago and Atlantic City, respectively.

TOXOPLASMOSIS

THE occurrence of cases of human toxoplasmosis in widely separated areas[1, 2] and a recent report of the results of tests for neutralizing antibodies in human serums[3] help to emphasize the increasing medical importance of this parasitic disease. The recognized types of toxoplasmosis include congenital encephalitis and hydrocephalus in infants, acute encephalitis in older children, acute febrile disease in adults resembling typhus or spotted fever and latent infection in adults detectable only by the presence of neutralizing antibodies in the serum. The clinical and laboratory diagnosis of such a protean disease poses an exacting problem.

One gains the impression from the published cases that the majority of infections are fatal, but several recoveries from acute infections have been reported.[4, 5] Moreover, the detection of neutralizing antibodies in the serums of mothers giving birth to infected infants[3] and also in the serums of persons in a random sample[6] suggests a carrier stage of the infection or recovery from an acute phase of the disease.

Although the clinical signs and symptoms in acute cases of toxoplasmosis are not specific, the outstanding basic lesions in the fetus, in infants and in older children are caused by the affinity of the parasites for the central nervous system. This involvement leads to hydrocephalus, cerebral calcification, encephalitis, mental retardation, convulsions and chorioretinitis. In adults, the parasites are more widely scattered in the viscera. Whether or not density of parasitization in adults determines the range of intensity of the infection from latency to acute fatal disease is not known. Nevertheless, an awareness of the inherent diagnostic difficulties in the different age groups is necessary if a specific diagnosis is to be made.

The occurrence of hydrocephalus, intracerebral calcification or chorioretinitis in children should suggest the possibility of toxoplasmosis. To verify the presumptive clinical diagnosis, serologic tests are available and animal tests can be performed with biopsy material, tissues, exudates and sputum.

Two serologic tests, the neutralization[7] and the complement-fixation[8] test, have been devised for the detection of antibodies to Toxoplasma. These tests are of value in determining both acute and latent infections. The neutralization test has been used most widely for surveys and for the diagnosis of acute infections. Although evidence has been presented to show that the absence of neutralizing antibodies in the serums of patients with chorioretinitis does not rule out toxoplasmic infection,[9] the test as used by the Minnesota Department of Health gave 163 positive reactions with serums from 761 patients.[3] The heat lability of the neutralizing antibody made it necessary for the Minnesota group to use serums stored under special conditions when fresh specimens could not be obtained. In the St. Louis survey, positive results were obtained with serums from persons giving no previous history of illness identifiable as toxoplasmosis.[6] The divergence of these results suggests some nonspecificity, and this possibility should be clarified to determine whether false-positive reactions occur with sufficient frequency to nullify the value of the test. Obviously this necessitates wide application in hospital and field surveys. The complement-fixation test, as devised by Warren and Sabin, appears of value only in acute infections, for the test becomes negative after the acute stage has subsided in experimental animals.

The inoculation of animals, particularly of white mice, guinea pigs and rabbits, with material from patients is of distinct value in attempting to isolate Toxoplasma.[10] These animals are highly susceptible

to this organism, and although suitable material for inoculation cannot always be obtained from patients, every effort should be made to isolate the organisms from patients with acute disease and with positive neutralization tests.

The serologic and animal-inoculation tests serve a twofold purpose: each extension of the former provides further evidence of their evaluation, and the development of toxoplasmosis in the animals serves to confirm the clinical diagnosis.

In the past, identification of the parasites in tissues has been difficult, primarily because of inadequate fixation and staining and because other parasites, such as Encephalitozoon, Sarcocystis and intracellular tissue stages of *Trypanosoma cruzi* and species of Leishmania, bear some morphologic resemblance to Toxoplasma. The use of proper fixatives[10, 11] and subsequent staining with polychrome stains yield films or sections in which Toxoplasma can be identified.

Although the epidemiology and mode of transmission of human toxoplasmosis have not been explained, a wide variety of wild and domestic animals and birds are susceptible to spontaneous infection with species of Toxoplasma.[12] Little is known about the specific relation of parasites from animal and human sources. Likewise, the role of animal infections as reservoirs for human infection is unknown: whether the parasites are transmissible mechanically from one host to another, by contamination of food and water or by the bite of arthropod vectors.

Since no drugs or antibiotics have been found that produce radical cures of acute infections due to Toxoplasma, chemotherapeutic studies are needed to search for a drug that will penetrate the host cells and destroy the parasites.

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MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Lowell	Sept. 5	Albert H. Brewster
Salem	Sept. 8	Paul W. Hugenberger
Greenfield	Sept. 8	Charles L. Sturdevant
Gardner (Worcester subclinic)	Sept. 9	John W. O'Meara
Haverhill	Sept. 10	William T. Green
Springfield	Sept. 16	Garry deN. Hough, Jr.
Pittsfield	Sept. 17	Frank A. Slowick
Brockton	Sept. 18	George W. Van Gorder
Worcester	Sept. 19	John W. O'Meara
Fall River	Sept. 22	David S. Grice
Hyannis	Sept. 25	Paul L. Norton

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments.

BOOK REVIEWS

Clinical Methods of Neuro-ophthalmologic Examination. By Alfred Kestenbaum, M.D. 8°, cloth, 384 pp. New York: Grune and Stratton, 1946. $6.75.

The book covers the field of clinical examination of the eye, the optic pathways and the associated muscles in a clear, orderly manner. The simple diagrams and tables are well integrated with the text. As a review of the clinical methods of ophthalmologic examination, the book can be highly recommended. For the clinic it will have to be used with other texts on cases and on experimental research.

A Manual of Tomography. By M. Weinbren, B.Sc., M.R.C.S. (Eng.), L.R.C.P. (Eng.), F.F.R. (Lond.), D.M.R.E. (Camb.). 4°, cloth, 270 pp., with 397 illustrations. London: H. K. Lewis and Company, Limited, 1946. Sh. 45.

Whether this technic is called tomography, laminagraphy, planigraphy, stratigraphy, vertigraphy or sectography is splitting hairs over historical priorities or the mechanical principles of the equipment with which one happens to be familiar. All these are forms of body-section radiography.

In the twenty-five years since Bocage patented three methods of moving the x-ray tube and the film reciprocally and in the seventeen years since Vallebonna published his work on the different principles of rotating the body of the patient on an axis between the tube and the film, it is perhaps strange that, except for McDougall's seventy-three-page monograph in 1940, no book dealing with the subject of body-section radiography has previously appeared. In this country the method has been utilized essentially in only a few centers, but abroad it has been embraced enthusiastically in many locations. Among the places where it has become most solidly established is Johannesburg, whence comes the present monograph by Weinbren. It is regrettable that this pioneer volume fails dismally to provide guidance to those who, given a little encouragement, might share the author's enthusiasm for the subject.

The book is presented more as an atlas than as a text. The mere printing of three hundred and ninety-seven illustrations on two hundred and seventy pages, however, does not automatically make it a reference book. The author is apparently well aware of this fact, and makes excuses for

himself while he places the responsibility for coming out in print on the editor of the *Clinical Proceedings of Capetown*. The material was evidently gathered together for a lecture given by the author in Capetown in 1944. The text, he admits, has been "cut too severely."

Nevertheless, the book establishes many of the good points of body-section radiography. In the chest field, the technic is seen to be particularly useful in delineating the bronchial tree without the introduction of iodized oil, in discovering cavitations within consolidated pulmonary tissue and in revealing details following thoracoplasty. It may be useful in determining heart sizes in cases in which the cardiac apex is obscured on conventional films by pleurisy or by an epicardial fat pad. Some portions of the spinal column are difficult to visualize on ordinary films, but even the cervicothoracic region, the lumbosacral articulations, the odontoid process and the atlanto-occipital joints may be shown in good detail on body-section films. Details of congenital scolioses, hemivertebras, fusions, spondylolyses and fractures may also be revealed. In the skull the method is particularly useful for delineating depressed fractures, the auditory canals, facial fractures and the temporomandibular joints. As extra embellishments, the author has used body-section radiography to outline the aorta in angiocardiograms, to observe the process of healing of fractures of the vertebral bodies, to distinguish traumatic Schmorl's nodes from those associated with adolescent kyphosis and to visualize excretory urograms in infants whose kidneys are obscured by intestinal gas.

For reference, the book is difficult to use because of the absence of an orderly sequence in its material. Only the briefest sketch of the historical development of the subject is included. A short chapter deals with the technic of filming various parts of the body, but one will search in vain for any mention of the mechanical principles involved in the apparatus. The book's most serious defect, however, is in the quality of reproductions, many of which are so poor as to put a considerable stretch on the imagination of the reader, prompted though he is by the captions.

Surgical Treatment of the Soft Tissues. Edited by Frederic W. Bancroft, M.D., and George H. Humphreys, II, M.D., Sc.D. Philadelphia: J. B. Lippincott Company, 1946. $15.00.

This book, which is an accompanying volume to *The Surgery of the Motor Skeletal System*, discusses injuries and deformities of the soft tissues. Hernia, superficial injuries, thermal injuries, infections, superficial neoplasms and circulatory disturbances are described by recognized authorities in these fields. The book is profusely illustrated, and each chapter has a well chosen up-to-date bibliography. This is a worthy addition to the previous volumes of this set.

NOTICES

METROPOLITAN STATE HOSPITAL

The Thirteenth Postgraduate Seminar in Neurology and Psychiatry will begin on Friday, October 10. The program will be as follows: a review course in basic neurology and psychiatry, consisting of eighty-one lectures, to be held every Friday from 2:00 to 10:00 p.m. from October 10 to December 12, 1947, and from January 2 to April 23, 1948, at the Metropolitan State Hospital, 475 Trapelo Road, Waltham; a course in social and special psychiatry, consisting of thirty-six lectures, to be held every Wednesday from 5:30 to 10:00 p.m. from October 15 to December 10, 1947, and from March 17 to May 12, 1948, at the Boston Psychopathic Hospital, 74 Fenwood Road, Boston; and a course in pediatric neuropsychiatry (child psychiatry), consisting of twenty lectures, the first two to be held on Tuesday, October 14 and 28, 1947, from 5:30 to 10:00 p.m. The remaining lectures of the course will be held on alternate Mondays from November 10 to December 8, 1947, and from March 15 to May 10, 1948, at the Walter E. Fernald State School, Waverley.

The seminar is open to all graduate physicians, but the number of vacancies for demonstrations in the review course in basic neurology and psychiatry will be limited to twenty-five in each of the subjects of the course: neuroanatomy, neuropathology and neuroroentgenology. If the number of applicants exceeds the available teaching facilities, preference will be given to physicians of the Department of Mental Health and of Veterans Administration hospitals and to those preparing for board examinations. Those interested are requested to apply in writing before October 6 to Dr. William C. Gaebler, superintendent, Metropolitan State Hospital, Waltham.

NEW ENGLAND SOCIETY OF ANESTHESIOLOGISTS

The first regular meeting of the New England Society of Anesthesiologists will be held in the Bigelow Amphitheater of the White Surgical Building, Massachusetts General Hospital, on Tuesday, September 16, at 8 p.m. The program will be as follows:

Operative Morbidity: A dual responsibility. Dr. Meyer Saklad, director of anesthesia, Rhode Island Hospital.

Report of Anesthesia Study Commission of the New England Society of Anesthesiologists.

NEW YORK ACADEMY OF SCIENCES

The list of speakers at the conference entitled "Antihistamine Agents in Allergy," which will be held at the New York Academy of Sciences on October 3 and 4, is as follows: Sir Henry H. Dale, formerly director of the National Institute for Medical Research, Mount Vernon House, England; Dr. Carl A. Dragstedt, professor of pharmacology, Northwestern University Medical School, Chicago; Dr. M. Rocha e Silva, professor of pharmacology and biochemistry, Instituto Biologica, Sao Paulo, Brazil; Dr. M. W. Chase, Rockefeller Institute, New York City; Dr. E. T. Waters, Banting and Best Department of Medical Research and Department of Physiology, University of Toronto, Toronto; Dr. N. Fell, Camp Detrick, Frederick, Maryland; Dr. D. Bovet, professor of pharmacology, University of Rome; Dr. Rudolf L. Mayer, Division of Research, Ciba Pharmaceutical Products, Incorporated, Summit, New Jersey; Dr. Earl R. Loew, Department of Pharmacology, University of Illinois College of Medicine, Chicago; Dr. Rolf Meier, medical director, Ciba Limited, Basel, Switzerland, and professor of pharmacology and therapeutics, University of Basel; Dr. Charles F. Code, Mayo Foundation, Rochester, Minnesota; and Dr. Samuel M. Feinberg, Department of Allergy, Northwestern University Medical School, Chicago.

Persons desiring to attend the conference may do so by making a request in writing to the executive secretary, Eunice Thomas Miner, Central Park West at Seventy-ninth Street, New York 24, N. Y.

NEW YORK ACADEMY OF MEDICINE

The Twentieth Graduate Fortnight of the New York Academy of Medicine will be held October 6 through October 17, the subject being "Disorders of Metabolism and the Endocrine Glands." The program includes morning panel discussions, afternoon clinics, evening lectures, scientific exhibits and demonstrations. Complete programs will be mailed to physicians on request.

A physician who is not a fellow of the Academy may obtain registration by sending his name and address, accompanied by a check for five dollars, to the Secretary of the Graduate Fortnight Committee, 2 East 103rd Street, New York 29, New York.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, SEPTEMBER 4

MONDAY, SEPTEMBER 8
*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

TUESDAY, SEPTEMBER 9
*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

WEDNESDAY, SEPTEMBER 10
*12:00 m. Grand Rounds and Clinicopathological Conference. (Children's Hospital.) Amphitheater, Peter Bent Brigham Hospital.

*Open to the medical profession.

(*Notices continued on page xiii*)